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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

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Ann Arbor, Michigan

Thirty-eighth Annual Meeting
THE AMERICAN ACADEMY OF PERIODONTOLOGY
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Miami, Florida

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The above picture was taken at the Cleveland Meeting of the Council of the American Academy of Periodontology and includes all members excepting Dr. Robert Kesel. They are, (standing left to right) Doctors Stanley Baker, Harvey Wood, J. Westbrook (retiring member), Joseph Bernier, Raymond Johnson (retiring member), Donald Kerr, Roy Elam (Chairman of Council), Harold Ray (President-elect). Seated, Clarke Chamberlain (Secretary), Mildred Dickerson, James Aiguier (Treasurer), Harry Lyons (President of the Academy). Academy member Dr. Howard Hartman took the photograph.

THE JOURNAL OF PERIODONTOLOGY

APRIL, NINETEEN HUNDRED FIFTY-FOUR

The Effects of Dietary Protein Deprivation and of Age on the Periodontal Tissues of the Rat and Spider Monkey*

by HENRY M. GOLDMAN, D.M.D., *Boston, Mass.*

THE possible relationship of dietary protein deprivation and changes occurring in the periodontium in animals has received but scant attention, although much research has been reported concerning other manifestations. The effects of protein deprivation on the periodontium have been described in the rat as alterations in the supporting and alveolar bone as well as in the periodontal membrane.[1] Root and alveolar bone resorption was reported by Hunter.[5] Recently, the effects on the periodontal tissues of young rats, after varying the protein content of the diet, were described as resulting in the animals' having smaller jaws and less osteocementum on the roots than the control animals.[3] Also, the interdental papillae were affected, and in total protein deprivation, there was a marked disturbance in the transseptal fibers. Osteoporosis of the alveolar and supporting bone became accentuated with increasing protein deprivation.

The present report deals with the changes in the periodontal tissues of albino rats and spider monkeys on a protein-free diet. This experiment is one of several conducted to ascertain whether a disease of the attachment apparatus—cementum, periodontal membrane, and alveolar bone—could be produced by a dietary protein alteration, whether age of the animal played a role in this disturbance, whether there was any selectivity in the skeleton system with respect to alterations in quality and quantity of bone tissue, and, lastly, whether there was any selectivity of location concerning these manifestations in the jaws themselves. The first and second phases of these experiments will be described.

MATERIAL AND METHODS

Sixty-one rats and 16 spider monkeys were used in the experiments to be described and were divided into groups on the basis of age. The rats were divided into two groups: 26 young animals, weighing approximately 150-200 grams, and 35 adult animals weighing approximately 300-350 grams. The monkeys were divided into two groups of 8, according to age. For each experiment control

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groups of 4 rats or 2 monkeys were included. The young monkeys had already shed their deciduous teeth and in two instances the deciduous teeth were still present. The adult monkeys had reached full maturity by at least three years.

The diet consisted of starch, 70%; nonnutritive fiber, 15%; vegetable oil (Hydrog.) 10%; salt mixture U.S.P. XIV, 4%; cod liver oil, 1%. Vitamin B-complex and ascorbic acid were added. This powder was mixed with water and for the monkeys was formed into round balls so that they could be picked up. The food was given in greater quantity than was eaten, the animals being allowed to eat ad libitum. The animals had free access to drinking water.

The rats were sacrificed at varying times depending upon the moribund condition of the animal (70-80 days). In some instances death had ensued. The monkeys were sacrificed under similar circumstances. These animals, however, lived longer than the rats (115-145 days). Complete autopsies were performed on the animals. The heads were dissected and cut sagittally in the midline and fixed in 10% formalin. The jaws as well as skeletal areas were radiographed. Histologic preparations were made, the jaws being decalcified in 5% nitric acid. The sections were stained with hemotoxylin and eosin, by Mallory's connective tissue stain and a modification of Wilder's silver stain, the latter being counter-stained with Harris's hemotoxylin.

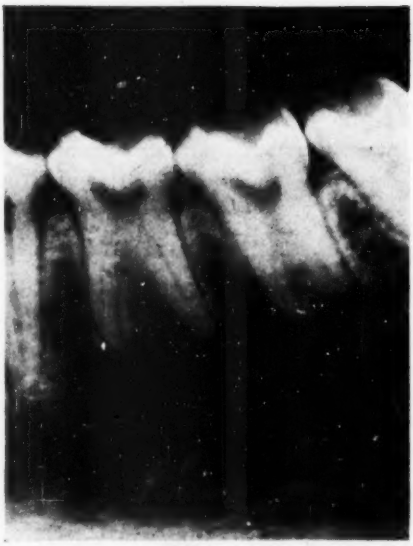

Fig. 1. Radiograph illustrating the mandibular molar region of an adult spider monkey used as a control. Note the thickness of the periodontal membrane space, the intact lamina dura around the teeth, and the dense cortical crestal bone of the alveolar process. The pattern of the trabeculae of the supporting bone in the jaw can be noted.

FINDINGS

A. Gross Findings

Rats: Both the young and old rats lost weight during the experiment. The hair in the young group became straggly and sparse, the skin appearing wrinkled and somewhat atrophic. The overall lack of size of the rat in comparison to the control was marked in the young group. The adult group was much less affected in this manner. Dentally, in the rat, no marked changes could be seen although in the young group in the molar region, a slight gingivitis was noticeable, the jaws were smaller than those of the control animals.

Monkeys: Although the animals could not be weighed accurately, weight loss was evident. Late in the experiment the animals became lethargic. Seemingly the young exhibited evidence of a slight enlargement of the abdominal area. Dentally there was some calculus formation and varying degrees of gingivitis were found. This gingivitis occurred in the form of an ulcerative necrotizing lesion. In isolated areas deep pocket formation could be detected. The teeth varied in mobility, some being very loose.

B. Radiographic Findings

Rats: In the rat there was a generalized radiolucency of the skull and the alveolar process in the molar regions as compared to the control animals. A slight exaggeration of the periodontal membrane space

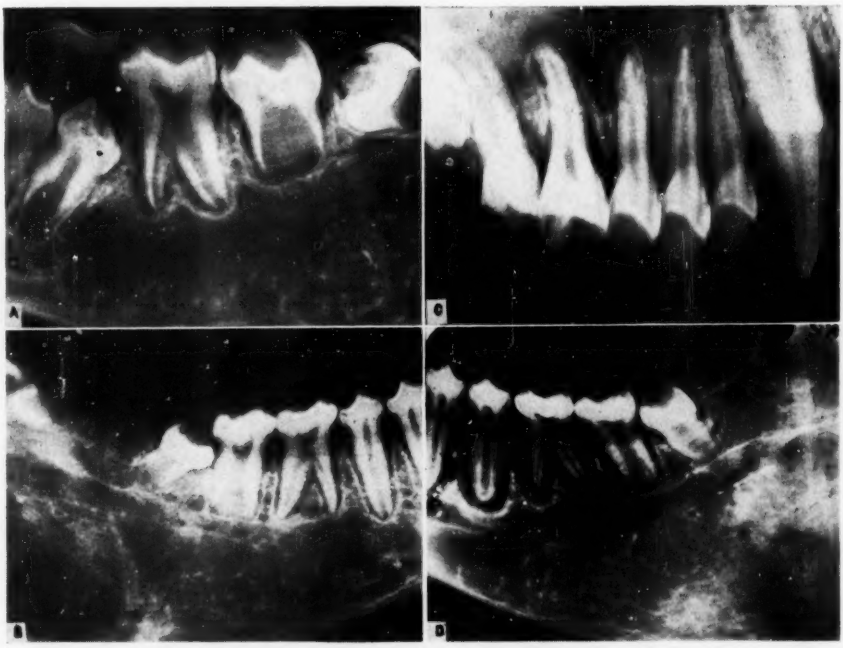

Fig. 2. Radiographs showing the effects of protein deprivation in the spider monkeys. In "A" the widening of the periodontal membrane space around the first and second molars is apparent. This is the radiograph of a young monkey, the deciduous teeth still being present. The radiopacity adjacent to the periodontal membrane space is increased in comparison to the norm. There is marked osteoporosis of the mandible. In "B" a radiograph of a young monkey, there is a marked widening of the periodontal membrane space. In this radiograph, also, marked osteoporosis of the mandible is evident. In "C," a radiograph of the premolar and molar maxillary region, the marked widening of the periodontal membrane space can be seen. On the mesial surface of the third premolar and first molar there is a distinct widening of the periodontal membrane space at the crestal portion evidenced by a diffuse rarefaction blending into the alveolar process. This is a characteristic radiographic lesion prior to loss of the alveolar crest. An advanced stage is illustrated in "D" in which the widening of the periodontal membrane space is extremely marked; the crestal bone is now resorbed and there is a diffuse osteoporosis of the body of the mandible. The radiopacity adjacent to the periodontal membrane space described in the text can be seen.

could be discerned. However, this latter feature could be assessed better after histologic preparations were compared with the radiographic observations. The adult rats showed distinctly lesser changes in these regards than did the young animals.

Monkeys: The radiographic changes in the young monkeys were especially of interest in that not only was there a distinct diffuse radiolucency throughout the skull, but there were characteristic changes observable in the alveolar and supporting bone. There was distinct widening of the periodontal membrane space long before any change in the crestal portion was observable. In fact, this manifestation seemingly preceded the radiolucent changes in the supporting bone. This widening of the periodontal membrane space may be seen in Fig. 2. Associated with this was a radiopacity of the alveolar bone which became more exaggerated as the disease process progressed; in fact, when the crestal portion had been completely destroyed, the radiopacity became more distinct. Close examination of this zone of increased density in the x-ray disclosed that it frayed out into the supporting area where there was a marked translucency. The interpretation of this radiographic feature could not be ascertained. In these animals, also, the

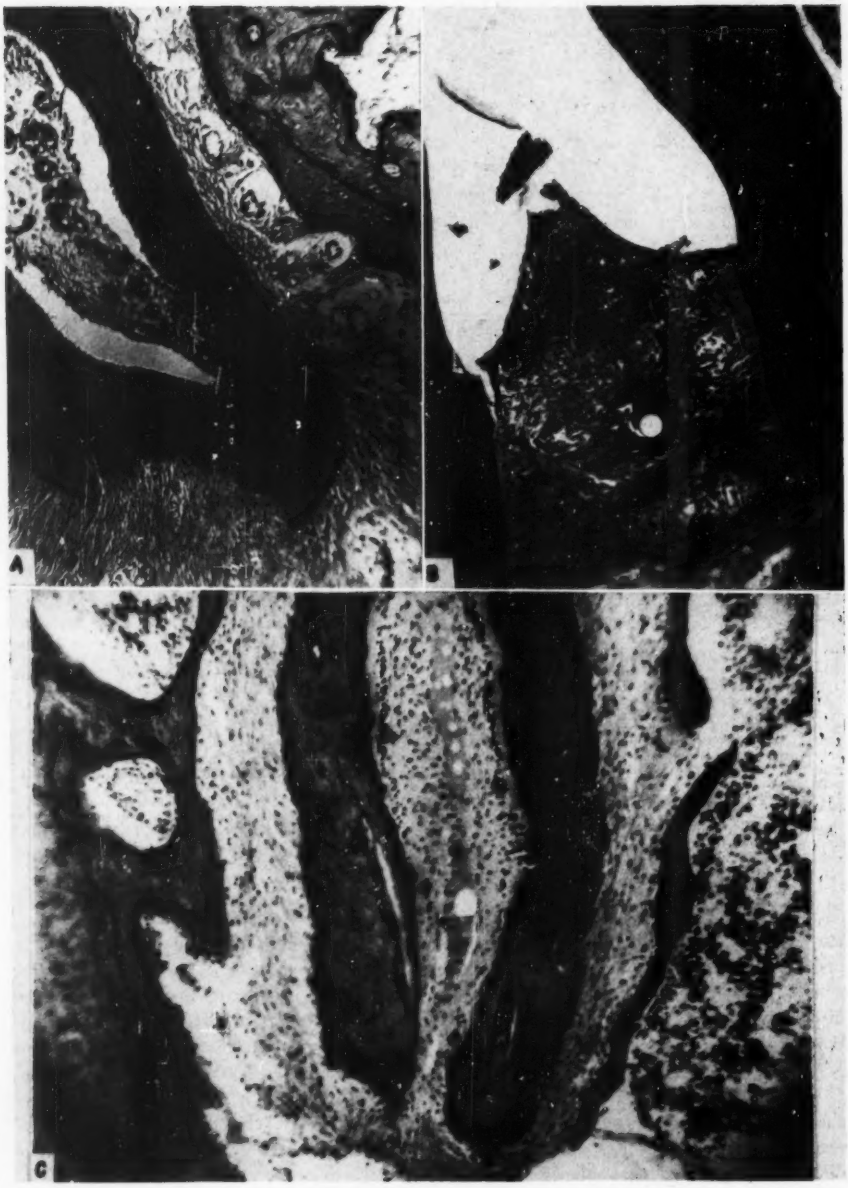

Fig. 3. Photomicrographs illustrating various histologic changes observable in the rat on total protein deprivation. Root resorption and changes in the periodontal membrane are seen in "A," inflammatory gingival changes with proliferation of the epithelium, and downward growth of the epithelial attachment are seen in "B," and the small amount of osteocementum of the roots of the teeth is seen in "C."

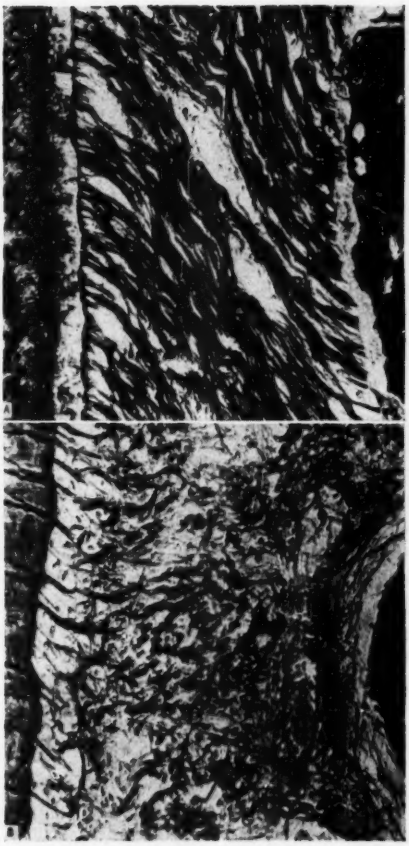

Fig. 4. High-power photomicrographs showing the alterations in the periodontal membrane in protein deficiency. The normal periodontal membrane (attachment apparatus) (A) may be contrasted to the manifestations seen in the deprivation animal (B). Note that the fiber apparatus is not only entirely disoriented but also has undergone degeneration. There is neither an osteoblastic nor a cementoblastic layer present, although remnants of a few fibers can be seen emanating from the cementum and the bone. (Modified Wilder Stain)

young showed distinct and exaggerated changes in comparison to the old group. The controls showed no evidence of any disturbance.

The changes in the long bones will be described in another report.

C. Histologic Findings

Rats: Histologic examination was conducted on the molar areas of the mandible and maxilla. Sections were prepared in a mesio-distal and a bucco-lingual plane. The interdental papilla showed evidence of changes in the fibers of the gingival corium. The fibers became blotchy in character, finally degenerating. An inflammatory infiltrate was seen, first a light sprinkling and later a heavy infiltration. The covering epithelium proliferated into the gingival corium and downgrowth of the epithelial attachment alongside the tooth surface was also observed. The transseptal fiber group was infiltrated by inflammatory cells and in some instances the epithelial proliferation from the gingival surface extended into the area. The late changes in the periodontal membrane were marked, the character and composition being entirely modified. There was a marked diminution of cells in the periodontal membrane, and concomitantly the principal fibers were reduced in number and in some areas even entirely lost and replaced by a loose, edematous tissue. Here and there a few indistinct intact principal fibers could be distinguished. The cementoblastic and osteoblastic layer of cells were for the most part completely destroyed, no cells as such, being discernible. The alveolar bone had lost its compact character as a lamina dura. There was a definite osteoporosis of the supporting bone with a tendency for a fibrous displacement of the marrow tissue. When considering the osteoporotic process, there was evidence that the resorptive process was active while bone apposition was almost completely absent.

Examination of the interdental bone showed evidence of resorption on the mesial surface; however, no bone apposition was apparent in the distal aspect of the septum. In some instances, resorption in this latter region was also observed. The alveolar crest showed loss of bone substance. As a rule, osteoporosis of the supporting bone was marked whereas the resorptive process in the alveolar bone was not as rapid. The supporting bone at a level of the tooth root exhibited large marrow spaces filled with hematopoietic tissue, only a few fragments of bone persisting in this region; a

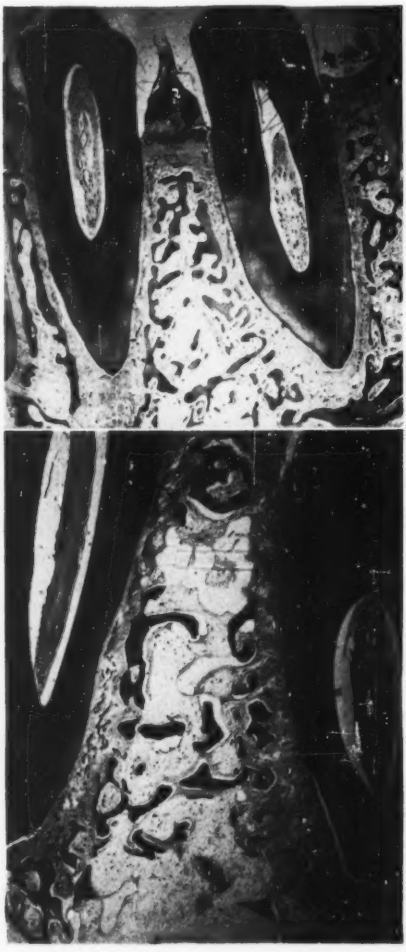

Fig. 5. Low-power photomicrograph showing the osteoporosis evident in the supporting bone in the premolar regions of two different animals. There are changes in the morphology of the periodontal membrane and the alveolar bone proper has been markedly altered.

resorptive process around these bone spicules was evident. No bone formation was apparent. Root resorption was seen in a few instances; this finding had been previously noted in protein deficient rats.[5]

Of extreme interest was a comparison of the above features in the young and middle and late adult animals. In the former, these changes were marked, whereas in the old animals, the described alterations were much less pronounced, only minimal lesions being noted. In one instance in an old animal practically no changes were evident in a time elapse longer than in comparable young animal.

Monkeys: Histologic examination of the jaws in the monkeys showed identical changes to those observed in the rats. Changes in the principal fiber apparatus of the periodontal membrane, and in the supporting and to a lesser extent in the alveolar bone, were apparent. Varying stages of alteration were noted. The collagen fibers in one area became blotchy in appearance while in another adjacent region they were missing. In a later stage an entire section could be seen which was void of almost all the collagen fibers running from the cementum to the alveolar bone, only short remnants being incorporated in the cementum. The spongy supporting bone in the earlier manifestations was considerably more affected than the cortical alveolar bone although the latter became completely altered in the later stages. Active osteoclasis as seen in the rat was not observed. For the most part, the bone trabeculae had an etched irregular surface consistent with Howship's lacunae with no observable osteoblastic layer evident on the opposite side.

The above findings may be best described by photomicrographs. Fig. 5 is a low-power photomicrograph of the premolar area showing marked widening of the periodontal membrane space and an osteoporosis of the supporting bone. The compact character of the normal alveolar bone has been completely disrupted, the tissue now being represented by irregularly shaped trabeculae (A). Gingival inflammation is marked and at "B" the papilla is destroyed. In Fig. 6 a high-power magnification of a section of the periodontal membrane, the character of the fiber apparatus is changed, both in the quality and quantity of the connective tissue fibers. Figs. 4 and 6 illustrate not only the changes in the perio-

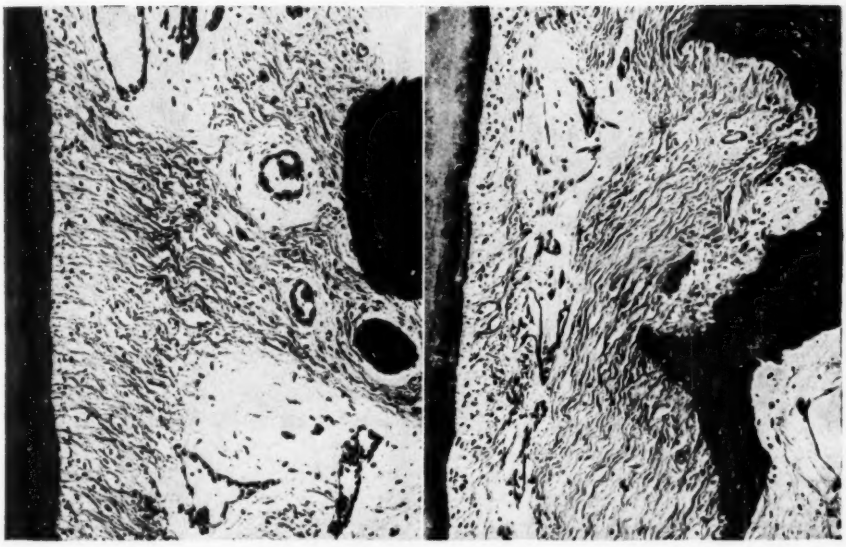

Fig. 6. High-power photomicrograph of sections seen in Fig. 5 showing the changes in the attachment apparatus. The periodontal membrane fibers are degenerating; some of the fibers, although poor in quality, are still present. The alveolar bone is undergoing resorption.

dontal membrane just described but also the resorption of the alveolar bone. Later changes in which the periodontal structures are completely disrupted may be seen in Fig. 8. The alveolar housing has been almost wholly destroyed as well as the gingival papillae. The periodontal membrane is completely disoriented, the tissue being now loose and edematous. Pocket formation may be seen at "P." A high-power photomicrograph of the character of the periodontal membrane is illustrated in Fig. 8, B and C.

As was noted in the rats, there was a marked difference in degree of the changes described between young and adult animals (Fig. 9).

DISCUSSION

Atrophic changes in the periodontal membrane and bone were the chief findings. Bone normally is constantly in a state of dynamic equilibrium, being formed while another portion is simultaneously being resorbed. It has been demonstrated that in order to form bone matrix, the protein metabolism must be normal. Apatite can be deposited only in the presence of an adequate matrix; when the synthesis of protein is insufficient, bone matrix is not formed. Thus, the replacement of bone substance does not take place. Seemingly, from this histologic study there was an increased rate of bone resorption; active osteoclasis may be demonstrated, especially in the rats. Also, the replacement factor was involved as well in that no new bone formation was apparent.

The turnover rates of collagen have been shown to be extremely low in comparison with those of intracellular proteins. This most likely holds true for the collagen fibers of the periodontal membrane. However, that there is a certain exchange rate for collagen has been demonstrated. Neuberger and Slack[9] found that administration of C^{14} labelled glycine, resulting in a high isotope content in the mixed proteins of liver and plasma leads to a relatively slight but definite C^{14} excess in the glycine residues of collagen of various tissues of the

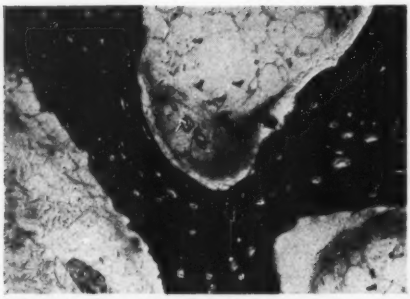

Fig. 7. A high-power photomicrograph of a trabeculae of bone seen in Fig. 5. Note the irregular surface of the bone denoting a resorptive lesion and the absence of an osteoblastic layer. The marrow is somewhat fibrosed.

rat. In rats more than 14 months old, the activities of the glycine samples obtained from all the collagen fractions were very low, the bone collagen, however, showing the highest values. In young adults, which were still growing slowly, low activity was observed with skin and tendon collagen, but appreciably higher values were obtained with collagen from bone and liver. In young, rapidly growing rats, all collagen fractions were high. The conclusions drawn from their experiments were that even in the adult rat, collagen fibers are continually, but very slowly, replaced, the turnover rates being highest for collagen from bone.

The alteration of the collagen fiber arrangement in the periodontal membrane, a principal feature of this experiment, was marked by final disappearance of the collagen. This change may be associated with either the normal turnover rate of collagen in relation to the time element of this experiment or may be the result of manifestation in the bone resulting in changes in the periodontal membrane because of the latter's dependence to both bone and cementum.

Ulceration of the interdental papilla described in the literature in rats, dogs, and monkeys as a result of various deficiency diseases, was also observed in this experiment.

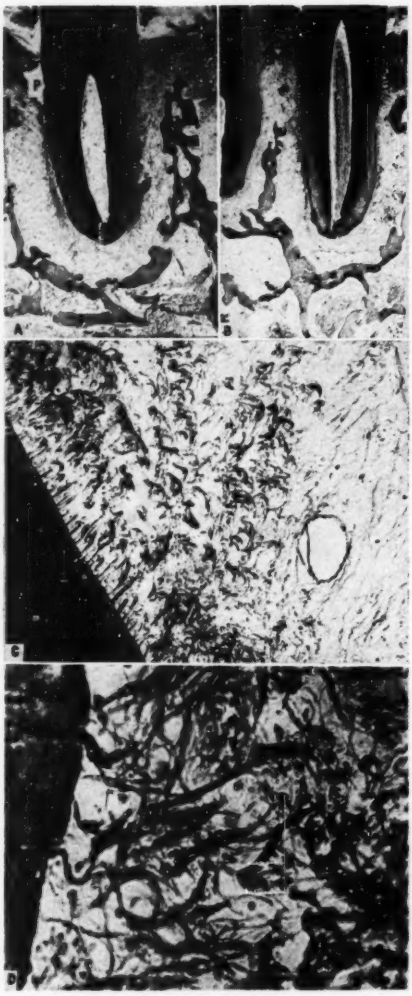

Fig. 8. Photomicrographs illustrating the changes in the attachment apparatus and alveolar process in the later stages. "A" and "B," low-power photomicrographs can be compared to Fig. 2D. Disorientation of the attachment apparatus and ulcerative necrotizing lesion of the gingiva are apparent. The alveolar process has undergone considerable resorption and in "B" interdentally it can be noted that the alveolar process has almost disappeared. The diffuse osteoporosis of the supporting bone can be seen. In "A" at "P" a deep pocket has developed. "C" shows a high-power photomicrograph of the attachment apparatus seen in "B," while in "D," the remnants of the fiber apparatus adjacent to the cementum are apparent.

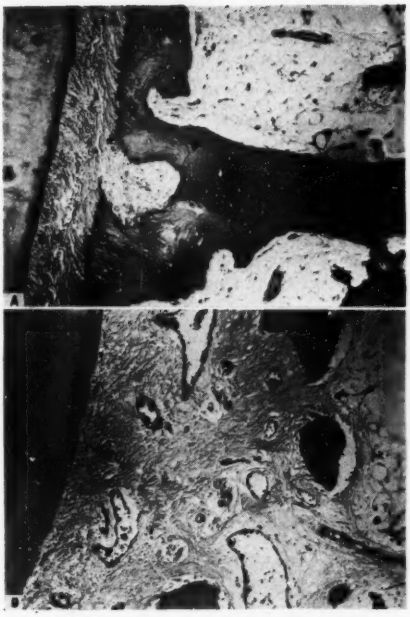

Fig. 9. The difference in severity of changes as seen in young and adult animals in the attachment apparatus is illustrated in the above photomicrographs. The severity of the lesion in the young (B) may be compared to that in the adult animal (A).

The contrast between the degenerative changes in the young and old animals was marked. Jaffe et al[6] explained such differences on the basis of an experiment in which hyperparathyroidism was produced experimentally in guinea pigs, rats, and dogs. They concluded that the characteristic lesion occurred in the bones of the most active growth irrespective of anatomical structure. This, they felt, explained not only the greater susceptibility of the bony skeleton of actively growing animals but also the quantitative differences in the response of different bones and of different portions of given bones. A similar explanation can be applied to this experiment. Also Neuberger and Slack in their study concluded that it seems likely that collagen including that of tendon and skin is metabolically more active in young rats than in adults. This, they believed, may be associated with the relatively large amounts of "procollagen" found in rapidly growing animals. Thus, the differences observed in changes in young and old animals may be attributed to a greater exchange rate of collagen found in the former.

SUMMARY

The effects of protein deprivation in young and old rats and spider monkeys resulted in degenerative changes in the attachment apparatus of the tooth. These findings are distinct from, and can be contrasted with, those conditions in which inflammatory marginal changes are induced by local etiologic factors, resulting in resorption of the crestal bone. These changes in the attachment apparatus due to protein deprivation consisted of the disappearance of fibroblasts and connective tissue fibers of the periodontal membrane, and the tissue's having a loose edematous appearance. Osteoporosis was evident, especially in the supporting bone; resorption of the alveolar bone occurred but was not as prominent as that seen in the supporting bone except in later changes where the alveolar plate also was completely altered.

The difference in the changes seen in young and late adult animals was marked, the young being severely affected in contrast with old animals. It is probable that the tendency towards degenerative changes resulting from systemic metabolic alteration is more severe in young animals because of the higher synthetic activity of their tissues as opposed to adult animals.

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Treatment of Solitary and Recurrent Oral Aphthae with Terramycin Mouthwash

by C. KENNETH COLLINGS, B.S., M.A., D.D.S.,* AND WALTER C. STOUT, D.D.S.,**
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ONE of the most discouraging problems faced by the dentist in his practice is the treatment of the various types of oral ulcers. Unfortunately the incidence of these ulcers is quite high and patients who are suffering from them are seeking relief in dental offices daily. They are really seeking relief from pain because these lesions, although usually not very large, are very distressing. In most cases, the dentist has been forced to meet this demand for relief chiefly with sympathy and assurances that the ulcers are self-limiting and will heal in time. Nothing has been completely effective in preventing their recurrence and very little success has been had in the past even in relieving the immediate pain being suffered by the patient when he comes for aid.

A great many medications have been tried for oral ulcers. A cure, as such, has never been found, but dentists, in an effort to give patients the immediate relief which they are seeking, have apparently tried almost everything available. A partial list of treatments that have been used would include bland mouthwashes, tincture of benzoin, 8% zinc chloride, camphorated phenol, spirits of camphor, Talbot's iodine, phenol, moccasin snake venom, systemically administered yeast, riboflavin, vitamin B complex, thiamin, radiation, methylene blue, gentian violet, aureomycin, sulfonamides, smallpox vaccine,[1,2,3] and influenza A & B virus vaccine.[4] An examination of the above list will show that the dentist has tried cautery, antiseptics, vitamins, radiations, sulfas, vaccines and antibiotics. Only moderate success has been attained with some of them and others have been merely empirical attempts to do something for a suffering patient.

Several investigators have reported various degrees of success in treating aphthae with aureomycin.[5,6,7,8,9] In some cases it was reported that relief of pain occurred in a short time and evidence has been offered that the duration and recurrence have been reduced. Pedler[10] reports the use of 15 mg.

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lozenges of terramycin in treating habitual oral aphthae. On eleven patients he found that the ulcers became painless and healed rapidly. Two of these were men who had a long history of recurrence of aphthae but were kept free of any lesions for a period of three months by continuous administration of five or fewer lozenges daily. He states that "Although no patients have been cured, the condition has been considerably modified by topical terramycin treatment."

Nathanson and Morin[11] have reported the use of terramycin systemically in the treatment of herpetic stomatitis and recurrent aphthous stomatitis. The effects obtained by them, however, cannot be entirely ascribed to terramycin since they were using it in conjunction with, and supplementary to, vitamin B and C therapy.

METHOD OF TREATMENT

This report concerns the use of terramycin in the form of a mouthwash for the control of the pain which occurs with oral aphthae. We were chiefly interested in the solitary and infrequent aphthae which presented less of a problem than the recurrent types and the Herpetic types. In the course of this work, however, we have treated a few patients who gave a history of recurring attacks of ulcers. In all cases, the results were the same.

After diagnosis and questioning of the patients on the previous history of similar attacks, their cooperation was requested for return visits for study. The patient was then given the required amount of terramycin and instructed in its use. All doses were dissolved in four tablespoonfuls (two ounces) of warm water. The patient was then told to take approximately one tablespoonful (one-fourth of the solution) and rinse the mouth thoroughly for 30 seconds *by the clock* and expectorate. The same procedure was to be followed for each of the remaining three tablespoonfuls of solution, thus giving a total rinsing time of two minutes for each treatment. Treatments were to be done at home after each meal and before bedtime. Patients were to return next day for examination and on such subsequent days as requested. Forms of terramycin used were the 250 mg. capsules, the 50 mg. soluble tablets, and terramycin for oral suspension, which is flavored. All quantities of terramycin, regardless of form, were dissolved in four tablespoonfuls of warm water. The 250 mg. capsules were opened and the contents emptied into the water.

RESULTS

This work was started in October, 1952 and by November, 1953, fifty-one patients had been treated. The first dosage used consisted of the contents of a 250 mg. capsule dissolved in the warm water. This was used on 16 patients. All patients in this group reported relief of pain in from 8 to 16 hours. Nine patients reported that all pain was gone by the next morning and the other seven reported great relief from pain but some soreness still present. In no case was the ulcer healed and in only one case could any obvious healing be detected by examination on the day following the initial treatment. The point of interest was that all were either painless or greatly relieved, although still present. These aphthae, when treatment was started, were from one day to five days old.

Since the terramycin from the capsules had a very unpleasant taste it was decided to try the terramycin for oral suspension. The next six patients were treated with 250 mg. of oral suspension terramycin in the warm water. Of these patients, three reported very little relief after three days, two reported some relief after two days and one failed to report back. From these few samples it would seem that terramycin for oral suspension, although tasting better, does not give the desired pain relief.

The next series of treatments was designed to determine if smaller doses would produce the same pain relief. Twenty-three patients have, therefore, been treated with

smaller doses, using the 50 mg. soluble tablet of terramycin as the unit. One patient was treated using only one tablet (50 mg.) in water. This patient was one we had treated three months previously using the 250 mg. capsule and, having gotten quick relief before, had come in for treatment of a recurrence. After use of the small dose now, this patient reported incomplete relief after two days. From this single case, it was decided to increase the dose to 100 mg. in each rinse.

The next twenty-two patients were treated with 100 mg. doses. Of these, fifteen reported complete relief from pain and seven reported definite improvement by the next morning. It would seem, from these data, that the 100 mg. dose is fully as effective as the 250 mg. dose.

Two patients, out of the forty-five treated, reported sensitivity to the terramycin. In both instances this sensitivity took the form of a relatively mild glossitis. The tongue was not swollen but appeared slightly red and mottled. Both patients were advised to return if the symptoms became worse. In both, the symptoms disappeared within a week without treatment. One reaction occurred after the use of the 250 mg. capsule and one after the use of the 100 mg. of soluble tablets.

Six patients have been treated using a placebo preparation. Four of these patients were receiving treatment from us for the first time. Two had received previous treatments from us using terramycin and, therefore, knew what to expect. No patient reported any alleviation of pain after using the placebo perparation for forty-eight hours or longer.

DISCUSSION

It is believed that the experiments described above offer to the dentist a quick, safe and easy method of relieving the pain which occurs with solitary aphthae and recurrent aphthae. In the thirty-eight cases treated either with terramycin from capsules or with the 100 mgs. of soluble tablets complete relief was experienced in twenty-four cases and most definite reduction of pain and soreness in the other fourteen cases. This relief occurred within 8 to 16 hours after treatment was started in all cases. Whether the ulcers were one day or several days old made no difference in the results.

No acceleration of healing rate was noted. The ulcers seemed to last as long as usual and healed in the usual manner. Also no evidence was found that the treatment prevented recurrence. Some of these patients have returned two or more times for our treatment, in each case receiving immediate and grateful relief but experiencing no reduction in the frequency of occurrence.

We have found only one other report suggesting an analgetic effect of terramycin when applied topically.[10] Perhaps this method of treating ulcers will prove of some value in the management of office patients who are in pain from aphthae. No reason can be given at this time to explain why terramycin relieves this pain. Nor can we explain why the more palatable oral suspension does not seem to ease the patient as the unpalatable tablets and powders do.

SUMMARY

1. A method of treating solitary and recurrent oral aphthae has been described, using a mouthwash of terramycin.

2. No evidence was found that terramycin used as a mouthwash decreased the frequency of recurrence of ulcers or hastened the healing of those present.

3. Evidence was found that terramycin used as a mouthwash is a potent factor in reducing or eliminating the acute pain present with these ulcers.

4. Seven or eight doses of 100 mgs. of terramycin, each dissolved in four tablespoonfuls of warm water, used p.c. and h.s. as described, seems to be the most economical dosage to secure the desired analgesia.

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Gingival and Periodontal Disease in Children

by WILLIAM G. MCINTOSH, D.D.S., *Toronto, Canada*

THIS paper discusses the above subject from three aspects:

1. How frequently do periodontal lesions occur in children of 6 to 11 years?

2. Will the patient 'grow-out' of periodontal lesions that are present at an early age?

3. How do the periodontal lesions of children compare microscopically with those in adults?

Clinical and microscopic evidence which help to answer these questions are presented.

The literature is full of controversial statements regarding the onset of periodontal disease. For example McCall[1] has written that the foundation of most periodontoclasia of adult life is laid in childhood. He points out that gingivitis is very common in children and considers it a phase in the development of periodontitis. Bunting[2] and also Broderick[3] and Brucker[4] reported that periodontal disease is seldom seen in childhood or youth—but it is confined almost always to persons over 25 to 35 years of age. Schour and Massler[5] reported that 40.2% of children in Italy showed signs of gingival disease. The same authors[6] concluded their report on the incidence of gingivitis in Chicago school children by saying that "attached gingivitis (periodontitis) was practically non existent in their group of children."

METHODS

Three hundred and ninety-eight children between the ages of 6 and 11 years were the subjects in a clinical study. They were all residents of a low-income section of Toronto. They were taking part in a Red Cross School Meal Study and had been

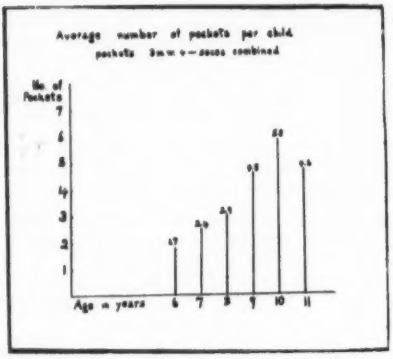

Chart 1.

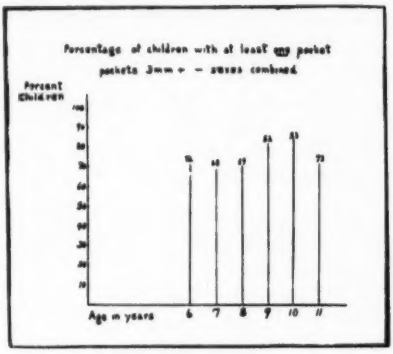

Chart 2.

selected as pairs for equivalent weight and height, etc. as reported elsewhere.[7] A full mouth examination for the presence of gingival sulci or pockets was made on these subjects using a William's calibrated periodontal probe. All pockets 3 millimeters or more in depth were recorded. The examinations were done by two examiners who saw different subjects and the results were totaled.

The microscopic material was procured from another source during routine autopsy examinations. The tissues secured were fixed in formalin decalcified with five percent nitric acid, blocked in celloidin and stained with haemotoxylin and eosin stain.

RESULTS

The number of children in each group was as follows:

6 years	25
7 years	83
8 years	79
9 years	84
10 years	88
11 years	39
Total	398

Chart 1 shows the average number of pockets (3 mm. or more in depth) per child. The average D.M.F. for the same 9, 10 and 11 year old children was 5.5. Hence there appears to be nearly as many early periodontal lesions as caries lesions in the same group of children.

Chart 2 shows the percentage of children in each age group who had at least one pocket. The average for all age groups is 74.5%.

If each tooth is considered to have four vertical surfaces it is possible to calculate the percentage of all surfaces with pockets in the maxilla and mandible. The results of these calculations for each age group are shown in Chart 3. With the exception of the eight year old children, all age groups had a slightly higher incidence of affected surfaces around the maxillary teeth than around the teeth in the mandible.

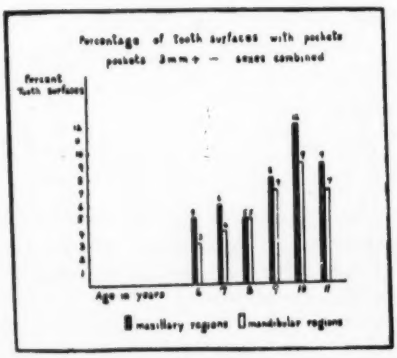

Chart 3.

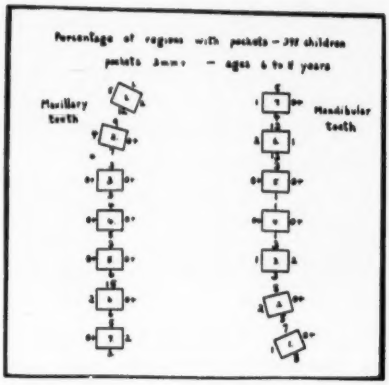

Chart 4.

Chart 4 shows in percentages the number of times each tooth surface was found to have a pocket when all age groups are combined. Both sides of the mouth are included in these figures. For example, the distal surface of the upper central was found to have a pocket 12 times in every hundred distal surface (right and left side) examinations. The numbers inside the squares indicate the tooth position in the arch. Numbers 4 and 5 represent either the deciduous molars or the first and second bicuspids depending on which teeth were in position at the time of examination. The greatest pocket frequency occurred at the mesial of the first molars around both upper and lower teeth.

In Chart 5 the pocket frequency for a specific location is plotted at each age level from 6 to 11 years. Two locations are used as illustrations. At age 6, approximately 15% of all upper first molars had pockets on the mesial surface. At age 11, approximately 25% of the mesial surfaces of upper first molars had pockets. At the labial surface of upper centrals, on the other hand, at 6 years of age about 10% had pockets. In the 11 year oid children the incidence at the labial of upper centrals was nearly zero.

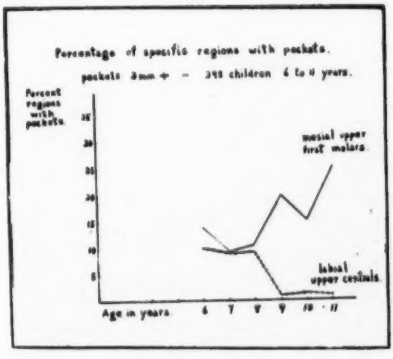

Chart 5.

Figure 1.

Figure 1 is a photomicrograph of a section which was taken four hours after death, from a six year old girl who died from a brain tumor. The photograph shows the ribbon-like remnants of the enamel cuticle with clusters of organisms attached (a). Point (b) represents the base of the gingival sulcus which is filled with organisms, desquamated cells and debris (c). The epithelium approximating the tooth is poorly stained and undergoing various stages of necrosis (d). The deeper epithelium has proliferated along the surface of the cementum (e). Inflammatory cells (f) appear in the area and are concentrated in the epithelium and connective tissue immediately apical to the base of the sulcus.

Figure 2.

Figure 2 is taken from a section procured from a six weeks old baby girl, three and a half hours after death by asphyxia. It shows one side of a tooth on which there was a sulcus similar to that shown in Figure 1. The base of the sulcus is above the border of this photograph. This picture is included to show the lesion in the vicinity of the bone crest (a) characterized by haemorrhage and vacuole formation (b), destruction of principal fibres (c) and loss of bone (d). Some infiltration of inflammatory cells can also be seen (e). Using serial sections this lesion was shown to be continuous with the gingival sulcus.

On the opposite side of the tooth shown in Figure 2 there was no gingival sulcus and the underlying tissues appeared healthy (Figure 3).

Figure 4 is a low power magnification of a gingival cleft (a) from the same six weeks old baby girl. The proliferation of the epithelium (b) into the underlying connective tissue can be clearly seen.

Figure 5 is a higher magnification of the area immediately below the open space (cleft) seen in Figure 4. It shows a tract (a), lined by epithelial cells (b) that have undergone some degree of keratinization, and filled with desquamated cells, organisms and cellular debris (c). No inflammatory cells were seen.

Figure 4.

Figure 3.

Figure 6 is from the area below that illustrated in Figure 5. The lateral dimension of the tract (a) is considerably reduced but it can be clearly seen penetrating into the deeper tissues and even cutting across an epithelial finger (b). The connective tissue fibres in the area immediately adjacent to the tract are indistinct. Few fibroblasts were seen in this area and again there was little evidence of cellular infiltration.

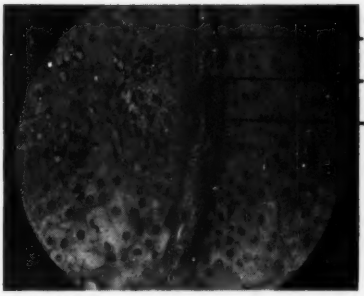

Figure 5.

DISCUSSION

While it is possible that some pockets were missed during the clinical examinations, those pockets that were recorded constituted an objective measurement about which there could be little doubt. No distinction was made between those pockets that were produced by loss of attachment between the soft tissues and the tooth and those that were produced by gingival enlargement (inflammation). However, any pocket that is three millimeters or more in depth provides a sheltered incubation zone in which debris, organisms and toxins can collect and from which destructive agents may attack the deeper tissues as was suggested in the microscopic sections. It seems very probable that these shallow pockets, regardless of type, may constitute an early stage of a more advanced periodontal lesion.

Figure 6.

Massler and Schour found in their survey of gingivitis in suburban Chicago school children that 80% of all gingivitis was exhibited around the anterior teeth. The figures in Chart 4 suggest that such is not the case for the distribution of pockets.

The gradual increase in pocket frequency around the mesial surfaces of upper first molars, shown in Chart 5, might be interpreted to indicate that pockets once formed in this area tend to persist and that new pockets continue to develop in this location in the older age groups. For the subjects participating in this study the pockets on the labial of upper centrals followed an opposite pattern. It therefore seems that some areas are more important than others from a treatment standpoint, not only because of a higher incidence of pockets, but also because in some locations the pockets appear to persist while in other areas they may decrease without treatment.

The method of clinical examination used in this study appears to be a practical one for obtaining evidence on the incidence and behavior of periodontal lesions. Other age groups should be included in further examinations.

The two types of periodontal lesion illustrated in the photomicrographs are typical of the inflammatory and necrotic lesions of adults described by Box.[8, 9, 10, 11] They are shown here to provide evidence that similar periodontal lesions may develop at an early age.

SUMMARY AND CONCLUSIONS

1. Using clinical and microscopic evidence periodontal pockets were shown to occur in children 6 to 11 years of age.

2. A method of assessing the incidence of periodontal diseases by recording incidence and depth of periodontal pockets has been suggested. The method appears to be useful in providing data about the inception and incidence of periodontal disease.

ACKNOWLEDGMENTS

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AMERICAN ACADEMY OF DENTAL MEDICINE ANNOUNCES ANNUAL MEETING

Diagnosis and Treatment Planning will be the theme of the Eighth Annual Meeting of the American Academy of Dental Medicine to be held at the Penn Sherwood Hotel in Philadelphia, Pa., on Friday, Saturday, and Sunday, May 14, 15 and 16, 1954. The program will include lectures and discussions on all aspects of the subject, by medical and dental authorities well-known in this phase of dental medicine.

A program of sightseeing and entertainment is also being arranged for those attending and their families. All members and interested dentists and physicians are cordially invited. Programs and reservations are available through the office of the National Secretary, Dr. William M. Greenhut, 124 East 84th Street, New York 28, N. Y.

Mitotic Activity of Fibroblasts After Damage to the Periodontal Membrane of Rat Molars*

by LUZ C. MACAPANPAN, JULIA MEYER, AND JOSEPH P. WEINMANN,** *Chicago, Illinois*

IN a previous report, the histologic changes following tooth movement in young rats have been described.[2] In these animals, the first, second, and third upper molars on one side of the jaw had been displaced by inserting a stretched piece of rubber dam between the first and second molars. Upon release of stretching, the elastic recoil of the sheet of rubber had led to an almost immediate widening of the adjacent periodontal spaces, and to corresponding compressions on the other side of the teeth.

The enlargement persisted and even grew more marked when the rubber sheet had been left in position for longer periods, in spite of the beginning apposition of bone. The widening extended along the whole depth of the periodontal membrane, and was somewhat more marked in the cervical than in the apical area. At the height of the displacement, the distance between the surface of the root and the alveolar wall was one and a half times that of the corresponding periodontal space on the other side of the jaw. The fiber bundles of the periodontal membrane remained attached to both tooth and bone and were stretched taut across the increased distance. There was hyperemia, but no inflammatory reaction, in the widened periodontal spaces.

The most surprising finding in this material had been the great frequency of mitotic figures among the connective tissue cells of the periodontal membrane.† Striking increases in mitotic frequencies seemed to occur particularly in sites of marked enlargement, and therefore it was thought that the cellular proliferation might be related to the repair processes then going on in the stretched collagenous fiber bundles.[2]

The periodontal membrane is a promising site for bringing out such a relationship if it exists, and for studying its quantitative aspects, because its anatomy makes it much easier than elsewhere in connective tissue to define experimentally equivalent volumes of tissue which permit rather precise comparisons between different experimental animals and between experimental and control sites.

In the present instance, the degree of displacement undergone by each of the molars could be established and compared to the increase in mitotic activity found in the widened periodontal spaces around the tooth. With this aim, a quantitative analysis of the changes in these two variables was undertaken. The present report deals with the results of this analysis.

MATERIAL AND METHODS

Information concerning the animals used and the preparation of histologic material has previously been given.[2]

The amount of displacement of the three molars and the increase in mitotic frequency in response to leaving a rubber sheet in position for periods varying from 1 to 72 hours were studied in 34 young mature male rats subdivided into nine experimental groups. Estimates of the degree of displacement and of the mitotic activity were made at the sites of greatest enlargement of the periodontal membrane, which were on the distal side of the first molar and the mesial sides of the second and third

*This investigation was carried out under contract No. W-49-007 MD-496 with the Medical Research and Development Board, Office of the Surgeon General of the United States Army and partially supported by the Dental Protective Association of the United States.

**Division of Oral Pathology, University of Illinois College of Dentistry.

†A similar observation was made by Kaare Reitan in 1951.[8]

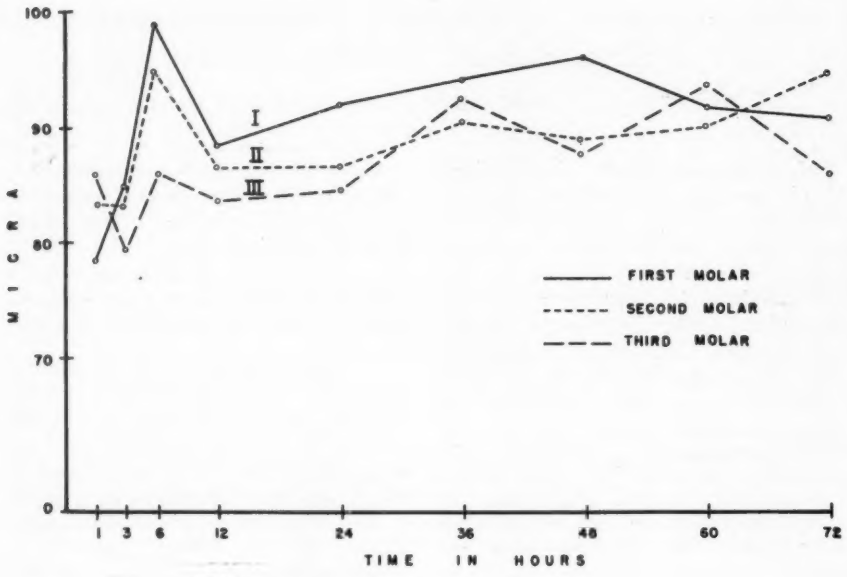

Fig. 1. Graph showing widths of periodontal spaces of control sides.

molars. For the sake of brevity, these sites will below be referred to as the periodontal spaces of the respective teeth.

1. Measurement of the degree of displacement of teeth at different experimental stages. The width of the periodontal membrane was measured at the cervical region of each root, where the alveolar crest provides an accurate landmark for locating corresponding levels in every root. For sections cut in the mesio-distal plane, the central section through each root was used. For sections cut in the horizontal plane, the section through the alveolar crest was used and the distance between tooth and bone measured along a straight line passing mesio-distally through the center of the root. Measurements were found reliable within 4-5%. The separate readings on the roots of each tooth were averaged, and this figure was considered as representing the width of the periodontal space of the tooth. The representative value for each experimental stage was obtained by averaging the widths in the four animals sacrificed at this stage.

To judge the degree of displacement which these widths represent, the same set of measurements was taken in the corresponding periodontal spaces on the other side of the jaw. Since, as will be seen, the experimental procedure also causes a transitory disturbance on the untouched side, the early experimental stages (1 to 6 hours) were compared to the control averages of the entire series. For comparisons following six hours, each experimental average was evaluated in terms of its own "control" value.

2. Increase in mitotic activity. All cells in mitosis in the quadrants used for measurement of width were counted. To make counts comparable, care was taken to include identical segments of each periodontal membrane. The sector in which counts were made was confined to the distance between alveolar crest and apex of root vertically, and to the area subtended

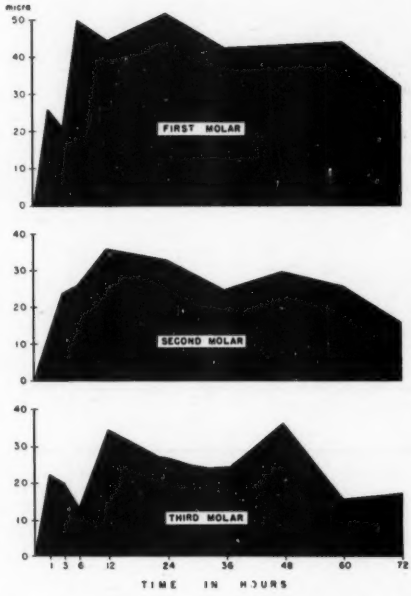

Fig. 2. Graphs showing the amount of displacement of the three molars.

by a 90° angle horizontally. This angle had its apex at the center of the root, and the line bisecting it passed through the center of the root in a mesio-distal direction. The counts for each root were added to give the count for the tooth. The total space in which counts were taken thus depends on the number of roots and on the length of each as well as on the width of the periodontal space.

Because variations due to the influence of the diurnal cycle on mitotic activity and due to the differences between individual animals had to be cancelled out, only the relative mitotic frequencies could be considered in this report. These were computed as the ratios of the experimental and control counts.

FINDINGS

1. Degree of displacement of the molars.

"Control side." Graph 1 shows the average width of the periodontal spaces on the non-experimental side of the jaw. That the experimental procedure has also caused a transitory disturbance on the untouched side can be seen from the fact that for all three molars the lowest value of the whole series occurs within the first three hours and is followed by the highest value within 6 hours. These early fluctuations are most marked for the first molar. At later experimental stages, there is only a slight degree of variation around the average. The average for these later groups is 92.0 $\pm$ 1.55 micra for the first molar, 89.3 $\pm$ 1.57 micra for the second molar, and 87.6 $\pm$ 1.95 micra for the third molar. The differences in width are not statistically significant.

Experimental side. Our measurements showed that the separation of the first and second molars enforced by the rubber dam had caused these and the subjacent tooth to be displaced by between 12 and 51 micra. The amount of displacement varied considerably from root to root, but of the 374 individual spaces measured, 371 were wider than the corresponding control ones, and when the root measurements for each tooth were averaged, every one of the 102 teeth had wider spaces than the control tooth.

The time course of the enlargement is shown in Graph 2. About a third to a half of the eventual displacement is seen to be established when the crowns have been kept apart for one hour. The effect of leaving the rubber dam in position for a longer time is considerable further displacement of all three teeth.

The three molars differ from one another in the degree and the timing of the displacement they undergo. For the second and third molars, the amount of displacement is the same and is only about two-thirds of that of the first (35 micra maximal shift as compared to 51 micra). For the first and second molars, on the other hand, the timing is similar and differs from the third. These two teeth show their maximal displacement centered around 15 hours,

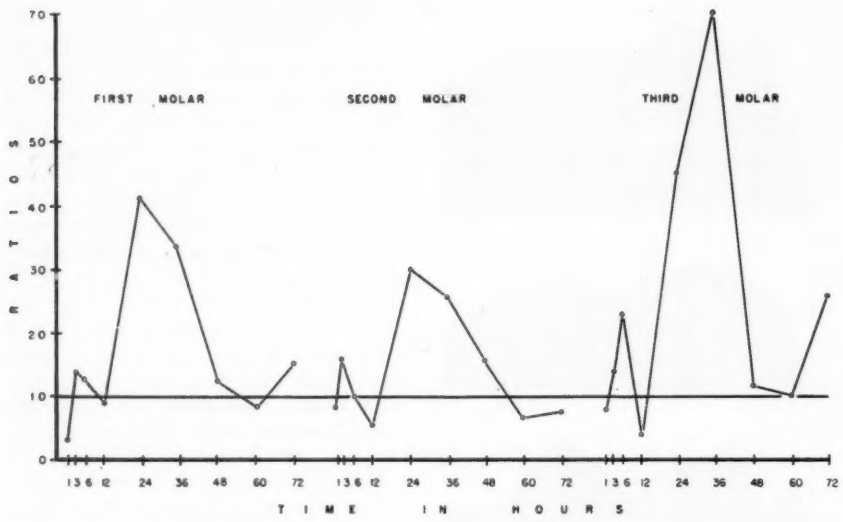

Fig. 3. Graph showing the ratios of mitotic frequencies of experimental and control sides.

the first molar being a little ahead of the second. Following 24 hours, the width of the periodontal space decreases somewhat, and to about the same degree in both teeth. During the early stages, particularly at six hours, the third molar shows less displacement than the second. By 12 hours, it reaches the same maximum as the second, but shows more erratic widths subsequently. By the end of the observational period, the periodontal spaces are considerably diminished in all three teeth.

The picture remains unchanged if instead of the absolute figures the percentage enlargement of the spaces is considered, because the periodontal widths of the three teeth on the control side are so closely alike.

2. *Mitotic activity.*

"Control" side. The total number of cells in mitosis was 1616. Of these, 927, or 27 per tooth, belonged to the first molar, 386, or 11 per tooth, to the second, and 303, or 9 per tooth, to the third. The difference between second and third molar corresponds to the difference in the number of roots of these teeth. The first molar probably has a higher mitotic frequency than the other two teeth. The animals were sacrificed in fairly even distribution over the hours of daylight (between 7 a.m. and 9 p.m.). Twenty-four hour figures including the nocturnal minima of mitotic activity would give somewhat lower averages. It is also possible that reverberations of the experimental disturbance caused some extra mitotic activity on the control side, particularly in the case of the first molar.

Experimental side. The total number of dividing cells on the experimental side was nearly twice that on the control side (Table 1). In all three teeth, the excess mitotic activity was almost entirely accounted for by increases in the 24 and 36 hour experimental periods. Mitoses in the seven animals making up these two periods amounted for the first molar to 68%, for the second to 61%, and for the third molar to 63% of the total count in all 34 animals.

The ratios of experimental counts over control counts are shown in Graph 3. For the third molar, reliable ratios could not be

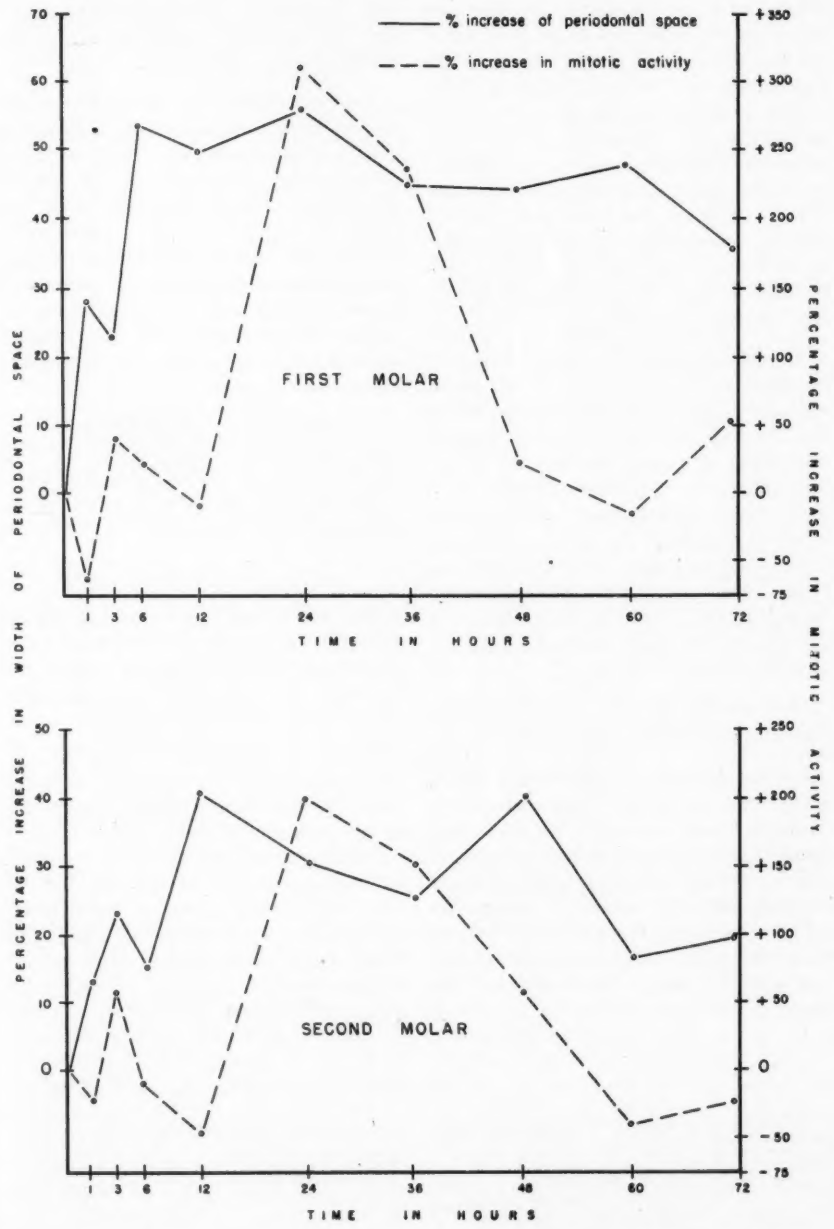

Fig. 4. Graphs showing the percentage increases in width of periodontal space and in mitotic activity.

obtained, because the absolute numbers of mitoses were too small. For the first and second molars, where all ratios were fairly reliable, control and experimental values were within 50% of one another in all instances except at 24 and 36 hours. At 24 hours, the mitotic activity of the first molar was four times and that of the second molar three times that of the control side. At 36 hours, the ratios were still 3.4 and 2.5 respectively. In the third molar, the ratios are erratic but the timing of the maximal activity is precisely like in the other two teeth (Graph 3).

All three teeth thus show a maximum of mitotic activity centered around 30 hours, and only minor deviations from normal at the other experimental times. A slight early suppression of mitotic activity due to injury may have occurred, as all three teeth show lower experimental than control values at one hour. Increased mitotic activity perhaps persists to some degree beyond 36 hours, as all three teeth are beyond normal at 48 hours. The main change in mitotic activity, however, is the great increase at 24 and 36 hours.

3. *Relationship between changes in periodontal width and in mitotic frequency.*

Percent increase in periodontal width and percent increase in mitotic frequency in the first and second molars are shown together in Graph 4. The marked enlargement of the first molar's periodontal spaces is associated with a marked increase in mitotic frequency, the smaller increase of the second molar with a slighter increase in mitotic frequency. The relation between the degrees of change in the two variables is in fact nearly numerically constant, at least for the peak periods of mitotic activity, as table 2 brings out.

Some conclusions can also be drawn concerning the timing of the two sequences of events, although there is a margin of uncertainty due to the intervals between observations. Maximal enlargement in the first molar is centered around 15 hours and is reached by 6. Maximal mitotic activity centers around 30 hours, the mitotic response lagging behind the widening by some 12 to 18 hours. These time relations are much the same in the second molar, and also in the third.

Widened periodontal spaces persist to some degree for at least 72 hours, and there is not much narrowing by 48 hours. Mitotic activity, on the other hand, returns to normal by 48 hours or shortly thereafter.

COMMENT AND CONCLUSIONS

The displacement of the molars did not cause bundles of principal fibers to be torn loose from the alveolar wall or root nor did it cause alterations of fibers that were visible in our histologic sections.

Abrupt displacement of a tooth to a degree obliterating the periodontal spaces ahead of the moving tooth is of course bound to lead to a severe degree of overstretching and damage to the fiber bundles in the wake of the movement. But the structure of these ligaments makes it unlikely that tearing of an entire bundle of fibers should occur or the injury become microscopically visible on hematoxylin-

TABLE 1.

Numbers of mitoses in the widened periodontal spaces and in the corresponding control spaces.

	Number per tooth, all experimental periods			*Total, all teeth*	*Number per tooth at peak of increase (24 and 36 hours)*			*% of peak count in total count*
	I	II	III		I	II	III	
Experimental	1692	630	669	2991	1153	383	423	65
Control	927	386	303	1616	313	138	83	33

TABLE 2.

Relation between changes in periodontal width and in mitotic frequency.

	Per cent increase in mitotic activity		*Per cent increase in width of periodontal space*		*Ratio of percentages*	
	I	II	I	II	I	II
24 hours	309.0	198.0	56.0	39.0	5.5	5.1
36 hours	235.0	151.0	45.0	27.0	5.2	5.6

eosin sections. The bundles of principal fibers, or periodontal ligaments, consist of sets of shorter fibers arising from the alveolar bone and cementum. In rapidly growing and erupting teeth, as for instance the incisors of the rodents, these sets of fibers are joined together in a rather prominent structure, the so-called plexus intermedius,[4] which occupies about the middle third of the periodontal membrane. This plexus is the site where the periodontal ligaments are continuously rearranged in adaptation to the continuous rapid changes in the relations of tooth and bone. In teeth of limited growth, such as the rat molars dealt with here, the relations between tooth and bone undergo equally continuous shifts, but these are much slower, and the site of junction of the fibers is a less conspicuous, indeed almost vestigial structure.

Forces within the physiological range as well as the excessive tension applied in the present experiments lead to alterations at this site.[1] The type of injury to be expected in the latter instance is a dissociation of individual fibers, rather than a tearing of an entire bundle of fibers.[5]

If it is reasonable to argue that similar degrees of displacement entail similar degrees of injury to fiber bundles, it may be concluded that the degree of cellular proliferation, which was found to correspond to the degree of displacement, also corresponds to the same degree of injury. This means that the degree of cellular proliferation corresponds to the magnitude of the necessary repair process. One would assume the cellular proliferation to be induced as part of the repair mechanism by which damaged fiber bundles are repaired or replaced and a widened periodontal membrane supplied with appropriately lengthened suspensory ligaments. A lasting enlargement of the periodontal spaces might result, but mitotic activity would return to normal once this is brought about.

SUMMARY

Mitotic activity of fibroblasts in the periodontal membrane was studied in 34 male rats of the Sprague-Dawley strain, 70 days of age, which had been subjected to experimental tooth movement. Shifting of the teeth which had led to enlargement of the periodontal space by from 35 to 55% on one side of the three molars examined was found to have led to increases in mitotic activity which varied with the degree of widening of the periodontal space. These increases followed the shifting of the teeth after an interval of about 18 hours, reached a maximum 24 to 36 hours after the beginning of the displacement, and dwindled to zero 48 hours after the initial displacement. At this time, and for another day, the periodontal spaces were still considerably, though no longer maximally enlarged. At the time of maximal mitotic activity, there was a nearly constant numerical relationship between the percent increase in mitotic activity and the percent widening of the periodontal spaces of the first and second molars. These findings suggest that fibroblast proliferation is part of the repair process by which the damaged fiber bundles of abruptly enlarged periodontal spaces are repaired and readapted to the changed relations between tooth and bone.

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Oral Tissue Reactions from Dilantin Medication in the Control of Epileptic Seizures*

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EPILEPSY has been known since ancient times. Descriptions of it are to be found in the Bible and in other early writings. To the condition have been ascribed such common names as "faints," "spells," "seizures," "fits," or "the sacred disease." Persons with epilepsy have been described as being "possessed with spirits." These empiric terminologies portray the more usually held concepts of the acute aspects of epilepsy, but do not delineate those transient episodes of sensory or psychic disorders which are parts of the total picture. Grinker and Bucy include this latter concept in their comprehensive descriptive statement of the syndrome—"recurrent paroxysms of convulsive movements, sensory, vegetative or psychic dysfunction with or without loss of consciousness." They conclude that this group of conditions comprises the "convulsive states" rather than a disease process. Preferably regarded therefore as symptoms of a disease, the different conditions which stimulate the aforementioned recurring convulsive seizures have been grouped together by Jackson and referred to as the "epilepsies."

These seizures are the consequence of some direct influence upon the central nervous system. The symptomatic epilepsies are promptly recognized and are produced by detectable disease processes. Intracranial birth trauma, arteriosclerosis, brain tumors, hypoglycemia, hemorrhage and embolism are a few of the etiological factors in these cases. Conversely, the idiopathic and cryptogenic epilepsies do not have easily discernible etiological factors. Ofttimes the etiology is never discovered. Grinker and Bucy insist that the symptomatic and idiopathic epilepsies have a common basis, referred to as the epileptic tendency or convulsive factor. Pathological and physiochemical brain changes are other factors which are "convulsogenic" in the presence of basic convulsive capacity. Congenital malformations of the brain, epidemic

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encephalitis, circulatory disturbances, multiple sclerosis, heredodegenerative processes, syphilis, tuberculosis and meningitis are abnormalities which Foerster lists as "irritative epileptogenous noxious agents." They produce pathological brain changes.

Many investigators have insisted that there exists a strong hereditary background in epileptic cases. It seems to be conceded by present-day authorities that a predisposition to epileptic seizures is partly on a hereditary basis because of the inheritance of certain characteristics in the individual nervous system. Mental deterioration may accompany the condition, but is not common today.

The seizures have been classified as major or "grand mal" attacks, and minor or "petit mal" attacks. In grand mal, a prodrome or aura precedes an impending convulsion. Premonitory symptoms are ordinarily in the form of some hallucination of the special senses of sight, taste or smell. The real onset of the convulsive attack is sudden and there is no relationship between it and the time or place of its occurrence. General characteristics of this type of seizure include loud utterances, rigor, clonic jerks, loss of consciousness, loss of control of micturition and defecation. These failures of psychosomatic and visceral coordinations frequently precipitate oral injuries. The convulsions terminate spontaneously and the epileptic is fatigued, stuporous, occasionally comatose and generally disorientated.

In petit mal, the manifestations are much less severe, being confined more or less to momentary spells of unconsciousness and an inability to continue with some current activity. Muscular movements are minimal.

Of especial interest to the dentist is the fact that in these seizures, the spasm may be so violent as to produce a dislocation of the temporomandibular joint. Frequently a tooth may be broken or dislodged (Fig. 1), and the patient may even fracture his jaw during the furious convulsive movements. Biting of the tongue and cheek, smacking, salivation, forcible chewing and swallowing are other symptoms of these "masticatory seizures," as they are frequently called. The persons affected are unconscious of what is happening, and may be totally unaware of the fact that the incident did truly occur. The spontaneous attacks may be initiated with a swallowing motion, followed by closure, traction of the mouth to the right or left and twitching of the lips. The duration of the seizure may be no more than twenty seconds. These masticatory seizures have their localization in the vicinity of one or other of the fissures of Sylvius. Chewing, smacking and salivation are due to activity of the lowest portion of the Rolandic cortex.

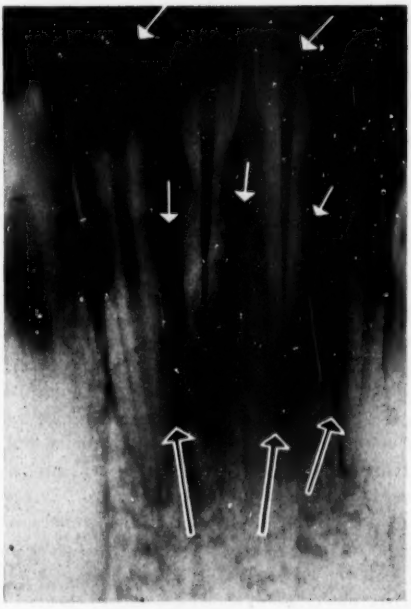


Fig. 1. Roentgenograms of lower anterior teeth 24 hours after masticatory seizure, violent enough to fracture incisal edges. No history of a fall accompanying seizure, but history of previous seizures. Note widening of periodontal membrane around the involved teeth and apical rarefaction.

The diagnosis of the epilepsies is based upon a careful observation of the seizures,

a detailed case history and encephalography. A determination can be made as to whether or not the symptoms are acquired. A differential diagnosis should be made between the epilepsies and the hysterias or malingering.

Epilepsy is widely prevalent. It has been estimated that 2 persons per 1,000 individuals in the population are in some way affected. Army statistics for World War I gave this number as 5 per 1,000. Selective Service figures from both World War I and II indicate that one out of every 200 persons has this disorder in some form. Conservative estimates place the number of epileptics in the United States as 500,000, with the occurrence in males and females being equal. Epilepsy is widespread among the various races of man. Several authors have claimed that the differences in incidence can be accounted for on the basis of race. More recently these apparent differences have been accounted for by other extraneous causes rather than by any particular racial predisposition.

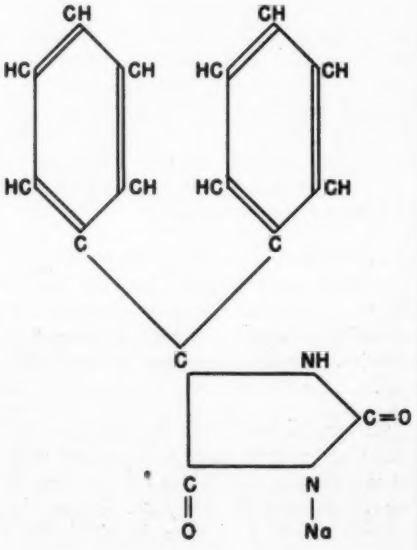
The chronic nature of the disorder, its wide prevalence and the relatively high cost of expert diagnosis, care and treatment are circumstances which emphatically indicate that epilepsy constitutes a problem of high order in public health and preventive medicine.

The success of treatment and favorability in prognosis of the epilepsies are directly proportional to the accuracy in diagnosis of etiology. Surgical removal of scars or irritable cortical lesions in acquired epilepsy is usually responsible for lasting benefits. The use of anticonvulsants and sedative medications is the most popular of current therapeutic procedures. Advocated also is a life free from emotional disturbances and critical decisions, since the latter tend to stimulate the onset of a seizure. The rationale of treatment of idiopathic epilepsy can be divided into (1) general hygienic, (2) dietary (utilization of ketogenic diets), (3) psychotherapeutic and (4) administration of drugs.

The drugs are used to control the quantity and intensity of convulsions. Among the commonly used drugs are the bromides, barbituric acid compounds and dilantin sodium. It is with the last mentioned of these anticonvulsants that the following account is concerned.

DILANTIN SODIUM

Dilantin Sodium or Diphenylhydantoin Sodium U. S. P. (Sodium 5, 5 Diphenyl hydantoin) or epanutin is an anticonvulsant drug which has no hypnotic action and is used to control the convulsions in epilepsy. It is analogous to the barbiturates and is a derivative of glycolyl urea. An odorless white powder with a bitter taste, dilantin sodium is soluble in water producing an alkaline solution with a hydrogen ion concentration of 11.7. It is often used in combination with phenobarbital. Ordinarily, the drug has a stimulating rather than a depressing effect. It is prepared in 30 and 100mg capsules (½ and 1½ grains), and has the chemical formula shown in the following diagram:



Structural formula Sodium Diphenyl Hydantoinate.

Fig. 2. Clinically normal pink gingivae.

Dilantin is more toxic than the bromides or barbituric acid compounds. The average daily dosage for adults is 4.5 grains, (0.1 grams three times per day). This beginning dosage may be increased to 3 grains (0.2 grams) three times a day. Children above six years of age are usually given 0.1 grams three times per day for one week after which the same amount may be given four times per day.

There are various side reactions which may occur from the use of dilantin Na. These include gastric irritation from the strong alkalinity, dizziness, dry skin, dermatitis, rash and itching, tremors, fever, nausea and vomiting, fatigue, apathy, difficult breathing and swallowing, nervousness, hallucinations and hyperplasia of the gingivae.

ORAL TISSUE REACTIONS

The most spectacular tissue reaction associated with the use of this anticonvulsant is exhibited in the gingivae. Normally, the gingivae are composed of the soft tissues which invest the cervical portions of the tooth and cover the alveolar bone. The gingivae have a dull stippled surface, fill the interdental spaces and continue in a smooth curve from one interproximal crest to the next. The color of the gingival tissues is variable and may range from a light pink appearance (Fig. 2) to a heavily pigmented purplish-black color (Fig. 3).

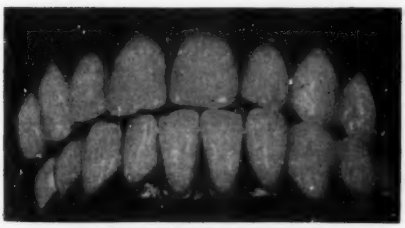


Fig. 3. Clinically normal heavy pigmented gingivae.

The administration of dilantin sodium produces an overgrowth of the gingivae. The gingival changes occur at variable periods of time after the first administration of the drug. These periods are, however, relatively short. The average period for the appearance of dilantin gingival changes is approximately three months. The method by which the drug exerts this proliferative reaction is at the present time, not known.

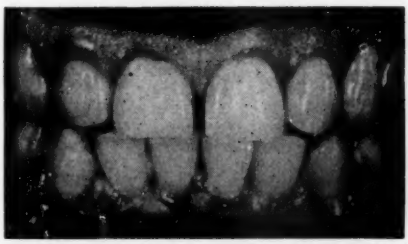


Fig. 4. Initial dilantin hyperplasia showing granular appearance, warty interdental papillae and beginning tissue cleft formations.

One of the first to record this hyperplasia was Kimball. In 68 of his cases he found varying degrees of hyperplasia. Fifty-seven per cent of patients using the drug for two or more months showed changes, while some who had taken it for eleven months showed no change. Kimball reported that the hyperplasia was suggestive of scurvy. A special investigation was conducted to determine whether or not dilantin inter-

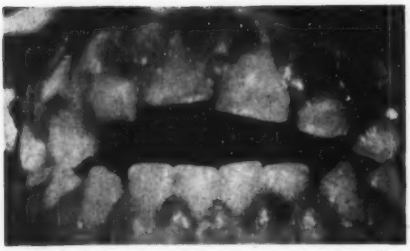


Fig. 5. Massive chronic gingival hyperplasia on labial and lingual teeth surfaces. Note non-occlusion of anterior teeth.

fered with ascorbic acid utilization. Animal experimentation indicated that the drug had no such influence. A study by Merritt and Foster concluded that gingival hyperplasia developing in patients undergoing dilantin therapy was not related either to ascorbic acid content of the plasma or to ascorbic acid utilization.

Beginning as a painless generalized non-inflammatory enlargement of the interdental papillae, the lesion later involves the remainder of the gingivae. An initial, stippled granular appearance is followed by a warty, lobulated growth with cleft formations (Fig. 4). Usually there are no subjective symptoms so that the condition is not brought to the attention of the patient. In early uncomplicated hyperplasias, the well-keratinized epithelium of the enlarged gingivae is protective and prevents pain and hemorrhage from moderately heavy masticatory use. If uncontrolled, the growth continues until it covers the labial, buccal and lingual surfaces of the teeth, eventually encroaching on the occlusal and incisal surfaces (Fig. 5). In many cases, mastication becomes difficult and painful procedure, if not an impossible process.

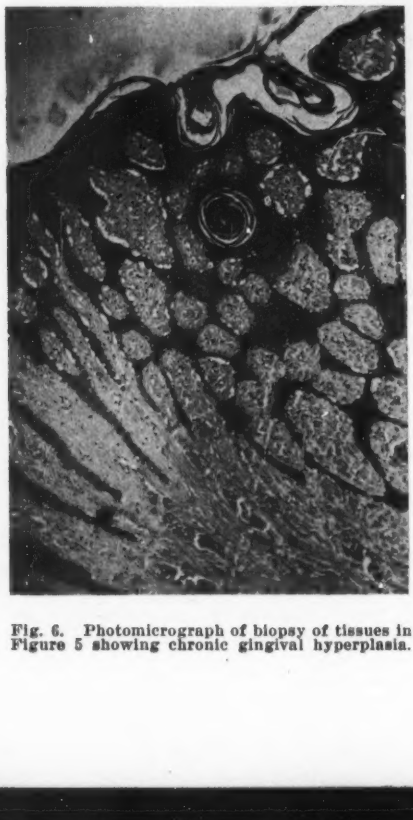


Fig. 6. Photomicrograph of biopsy of tissues in Figure 5 showing chronic gingival hyperplasia.

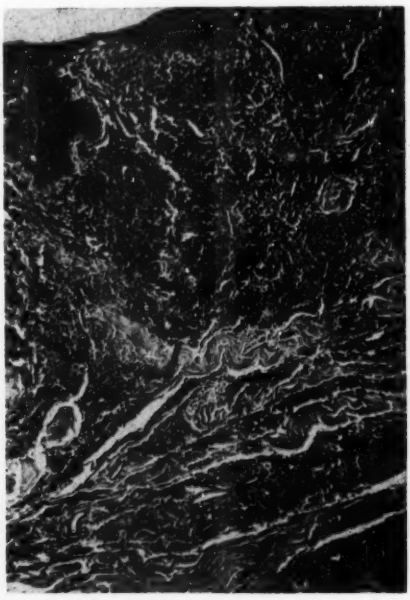


Fig. 7. Photomicrograph of chronic hyperplasia complicated by gingival ulceration.

HISTOPATHOGENESIS

Microscopic examination of hyperplas-

tic gingival tissue resulting from dilantin sodium administration shows acanthosis of the epithelium and downgrowth of the epithelial rete. Occasionally there is pearl formation. The corium shows edema of the papillary layer and increase of the collagen fibers of the submucosa with bone heteroplasia. Figure 6 is a histopathologic section of the tissues shown in Figure 5. The epithelium presents a moderate degree of hyperplasia with fine finger like anastomosing extensions into the underlying connective tissue, forming epithelial pearls and rete pegs. The connective tissue consists of interlacing bundles of collagen. There is a slight infiltration of chronic inflammatory

Fig. 8. Clinical picture of tissues shown in Figure 7.

cells in the connective tissue immediately subjacent to the epithelium. Lymphocyte and plasma cell infiltration is a secondary manifestation. This patient had been receiving dilantin for four years.

Figure 7 illustrates a histopathologic section of the gingival tissue depicting focal ulceration. There are necrotic destruction of mucosa and massive mononuclear infiltration of the connective tissue stroma. In this case the gingival hyperplasia is complicated by ulceration of the gingival margins. The patient has been receiving dilantin for three years. Clinically, there is evidenced gross oral neglect (Fig. 8).

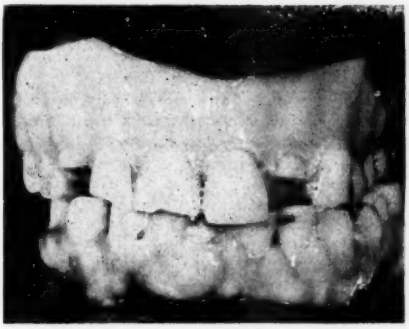


Fig. 9. Oral sepsis, periodontal disease, faulty prothesis with hyperplasia of tissues in lower jaw.

CASE HISTORIES

The following case histories have been selected to illustrate the various factors which may or may not influence the quality or quantity of hyperplasia.

CASE 1. P. N., Negro, World War I veteran, 56 years of age, had been hospitalized for ten years. He experienced seizures twice monthly. His medical diagnosis was epilepsy grand mal with mental deterioration. He was receiving 1½ grains of dilantin sodium three times per day since 1944. Oral examination revealed: (1) missing teeth all uppers. 17, 30, 31, 32, (2) moderate oral sepsis, (3) unserviceable upper denture with no retention and fractured teeth, (4) roentgenographs showed extensive bone resorption around lower incisors, widening of the periodontal membrane space and pocket formation, (5) firm, resilient, bluish pink, lobulated, hyperplastic gingivae around lower incisors (Fig. 9). Histopathology: Epithelial hyperplasia with superficial layers undergoing keratinization and focal exfoliation. Rete pegs penetrate underlying connective tissue. No evidence of inflammatory reaction (Fig. 10).

CASE 2. J. M., Negro, World War I veteran, 46 years of age, experienced dizzy spells and headaches since 1943. Later he developed convulsive seizures with tongue biting and fainting. His medical diagnosis was epilepsy grand mal, with moderate mental deterioration and marked psychomotor retardation. He had been receiving dilantin sodium 1½ grains (b.i.d.) since 1949. Oral examination revealed: (1) missing teeth—1, 14, 18, 19, 30, 31, 32, (2) extreme oral sepsis, (3) dental caries, (4) advanced gingival hyperplasia with enlargement of interdental papillae, warty and granular gingival surface, lobulation and cleft formation (Fig. 11).

CASE 3. J. G., Negro, World War I veteran, 62 years of age, was admitted to hospital in 1946 for treatment of hemiplegia, right face, arm, leg and motor aphasia. His medical diagnosis was hemiplegia, hypertensive arteriosclerotic encephalopathy with epileptiform seizures, motor aphasia. He experienced numerous epileptiform seizures, probably due to hypertensive, irritative organic basis. He had been receiving dilantin sodium 1½ grains (b.i.d.) since 1947. Oral examination revealed: (1) missing teeth—1, 18, 20, 29, 31, (2) unserviceable crown on 8, (3) occlusal trauma, (4) extreme oral sepsis, calculus, materia alba (inability to open well, probable factor in lack of hygiene), (5) absence of gingival hyperplasia (Fig. 12).

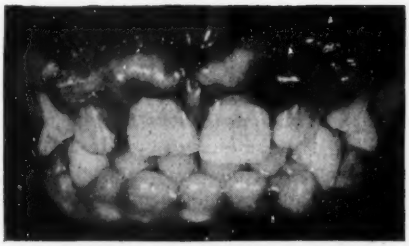


Fig. 11. Oral sepsis and dental caries with generalized warty hyperplasia, lobulation and cleft formation.

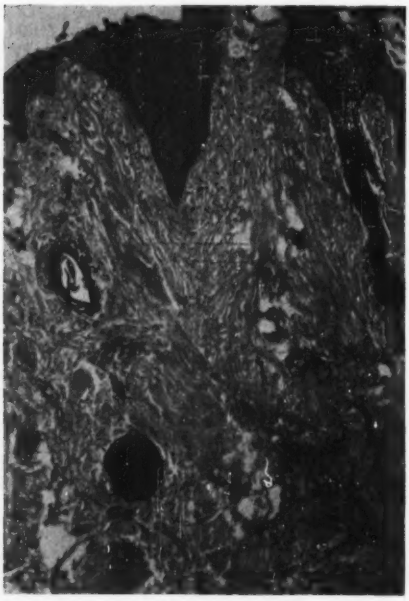


Fig. 10. Photomicrograph of biopsy of tissues in Figure 9 showing epithelial hyperplasia of gingivae.

CASE 4. J. R., Negro, World War I veteran, 55 years of age. Epilepsy always present. His medical diagnosis was epilepsy, idiopathic, grand mal with psychotic reaction. Dilantin sodium 1½ grains (b.i.d.) since 1941. Oral examination revealed: (1) completely edentulous, firm ridges, (2) no evidence of hyperplasia (Fig. 13).

CASE 5. W. P., Negro, World War I veteran, 58 years of age, hospitalized fourteen years with frequent episodes. His medical diagnosis was epilepsy grand mal with gross mental deterioration. Dilantin sodium 1½ grains (t.i.d.) since hospitalization. Oral examination revealed: (1) edentulous ridges, (2) labial and lingual surfaces of anterior ridges have several raised, warty, stippled areas (Fig. 14). The time which had elapsed since extraction of the teeth was not known. A biopsy of one of the raised areas showed areas of epithelial inclusion in connective tissue stroma. Foci represent epithelial digitations from basal mucosa. Occasional focus of mononuclear infiltration in stroma of no pathologic import (Fig. 15). Diagnosis was made of normal gingiva.

CASE 6. J. H., Negro, World War II veteran, 25 years of age, was admitted to hospital in 1949. Emotionally unstable, irascible, temper tantrums in Armed Forces resulting in discharge. His medical diagnosis was epilepsy, grand mal, idiopathic with epileptic personality. Dilantin sodium 1½ grains (b.i.d.) since 1949. Oral examination revealed: (1) missing teeth—5, 31, (2) relative oral cleanliness, (3) malocclusion, (4) mild, generalized hyperplasia with festooning and clefts around lower anterior teeth. Increased stippling and granular appearance of tissue surface.

CASE 7. R. W., Negro, World War II veteran, 45 years of age. Epileptic seizures, three or four per month since 1945. Onset marked by hot sensations. His medical diagnosis was psychoneurosis,

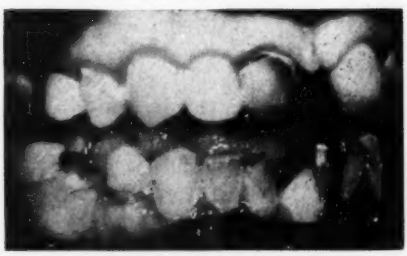


Fig. 12. Oral sepsis, calculus, mechanical irritation, occlusal trauma without hyperplasia.

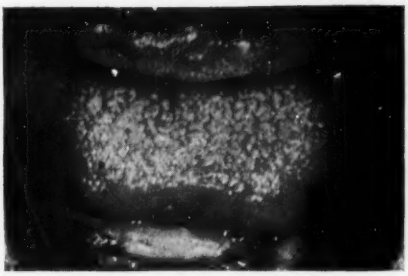


Fig. 13. Edentulous ridges with no hyperplasia.

conversion reaction, epileptiform seizures, mild mental incompetence. Dilantin sodium 1½ grains (b.i.d.) since 1946. Oral examination revealed: (1) missing teeth—1, 5, 6, 13, 19, 20, 30, (2) oral cleanliness, serviceable restorations, (3) moderate abrasion on remaining teeth, (4) relative absence of gingival hyperplasia.

COMMENT

The foregoing histories have been selected from a number of cases of epileptic patients at the Veterans Hospital in Tuskegee, Alabama. This hospital has a total of 1700 beds designed specifically for patients with psychiatric and neurological ills. There were approximately 150 Negro male patients diagnosed as epileptics at the time of this survey. It was possible to make oral examinations of 123 epileptics, 47 of whom showed varying degrees of gingival tissue transformations.

Twenty-four (24) exhibited what may be regarded as a typically severe hyperplasia which extended over portions of the clinical tooth crown. No other oral tissues except the gingivae were affected by the hyperplasia. The results of this survey do not tend to support frequent unsubstantiated claims relative to racial determinants of incidence. It would appear that like age, sex, weight and height of the patient, race does not influence the incidence of gingival reaction.

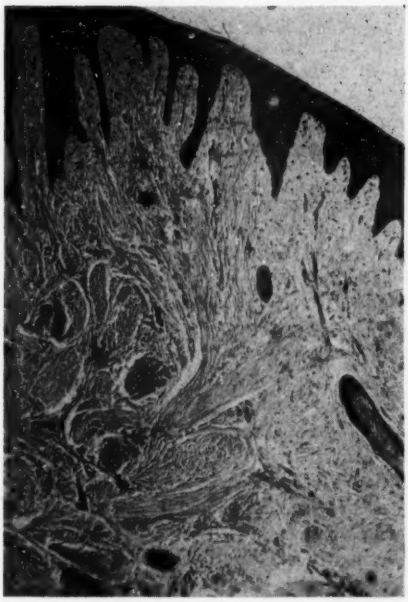


Fig. 15. Photomicrograph of biopsy of tissues in Figure 15 showing normal character.

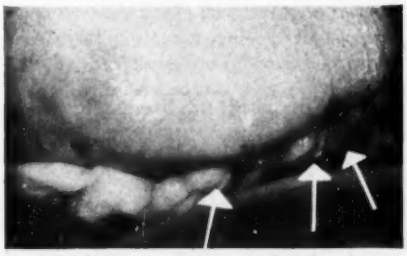


Fig. 14. Edentulous ridges with several raised pseudo-hyperplastic areas on labial and lingual ridges.

The wide variations in clinical manifestations demonstrated by the cases selected for this survey emphasize the practically inescapable fact that patent gingival reactions are dependent more or less upon an individual factor, which has been designated diversely as an intolerance, a sensitivity, an allergy or an idiosyncrasy to the drug. Too, it has been said that certain persons possessed a basic tendency to proliferative reactions.

Several investigators have indicated that the amount, the shape, color or consistency of dilantin hyperplasia may be influenced by such factors as oral sepsis, mechanical irritants, malalignment of the teeth, non-occlusion, occlusal trauma and the presence of varying degrees and types of periodontal disease. Many of these factors are frequently responsible for the inflammatory changes in the gingival tissues which become superimposed upon the initial dense, insensitive and resilient hyperplasia. Their role as primary etiologic factors in initiating gingival hyperplasia however, has not been substantiated by clinical or histopathologic observations.

Of great interest is the relative absence of clinical hyperplasia in Fig. 12 where the amount of irritation from poorly constructed crowns, the large aggregate of materia alba, the supragingival calculus and sordes are much in excess of the oral debris and accumulations to be seen in Figures 9 and 11, two of the cases which display a super-abundant overgrowth.

This supports the conclusion that the initiation of the hyperplasia is not produced by the irritating effects of prosthetic appliances. Irritation may intensify the reaction when there is an already existing tendency. Included among the factors which induce mechanical irritation are overhanging fillings, faulty margins of crowns and bridges and calculus. The impingement of these chronic irritants upon the soft tissues produces gingivitis and additional hyperplasia. Hemorrhage may be an attendant symptom in these cases.

The absence of hyperplastic tissues in edentulous mouths of patients undergoing dilantin sodium therapy is of interest. The case in Fig. 14 emphasizes the necessity for caution in specifying tissues as hyperplastic. This clinical appearance of hyperplasia in an edentulous case was not corroborated by histologic analysis. In Fig. 9, there was hyperplasia in the lower anterior region around existing teeth, but there was none in any areas of the upper edentulous arch. Nor was there any evidence of demonstrated pathological hyperplasia on the ridges in edentulous areas of any of this group of patients. In this respect then, there would appear to be general agreement that gingival hyperplasia, like other forms of periodontal disease, requires the presence of teeth in order to occur.

Habitually stimulating inflammatory changes, inadequate oral hygiene which accompanies malalignment of the teeth, may also influence gingival enlargement. Particularly is this seen when the anterior teeth are affected. The color of pink gingival tissues is altered to a dusky blue pigmentation while there are no appreciable color changes in highly pigmented tissues. Glickman attributes the color changes to venous stasis and intra-tissue hemorrhage.

The hyperplasia is greatest in the anterior region of Fig. 5 where there is a non-occlusion of the incisors and cuspids.

From a consideration of the cases in this survey, it would appear that oral sepsis, various forms of mechanical irritation, malalignment and non-occlusion do have some aggravating effect on hyperplasia when the latter occurs as a result of the administration of dilantin sodium. Whether or not various forms of periodontal disease or other local etiologic factors of periodontal disease have similar effects, is still controversial.

The elimination or correction of contributing factors is one of the essentials of therapy. Withdrawal of the drug is one method of treatment. Diminution of the dosage is preferable to complete elimination, and several cases have been benefitted considerably in this manner. Normally this infers an immediate cessation of further enlargement, rather than a subsidence of the hyperplasia.

Genuine abatement of the overgrowth can be produced by conservative periodontal treatment, which is recommended in the majority of cases and includes maintenance of scrupulous oral cleanliness,

vigorous stimulation by massage and toothbrushing, elimination of mechanical irritants and home care. If the hyperplasia is resistive and unsightly, tissue excision is advocated. The hyperplasia tends to recur promptly after treatment unless the contributing factors are eliminated and home care is maintained. As previously discussed, the overgrowth disappears entirely if the teeth are removed, even though dilantin medication is maintained.

One of the newer anti-convulsant drugs which has been employed in the treatment of elipepsy is mesantoin. It is a hydantoin (3 methyl 5,5 phenylethyl hydantoin) and has been prescribed in the treatment of generalized convulsions. Caution is advocated because of the possibility of blood dyscrasias which have been reported following its use. Other side effects such as gingival hyperplasia have not been observed following the prolonged administration of mesantoin.

Tridione (3,5,5, trimethyl oxazolidine 2,4 dione) is another anticonvulsant which has been found to be efficacious in treating some cases of petit mal. No oral tissue effects have been reported following its administration. Both of these newer drugs are not regarded as being superior to dilantin which, despite its oral tissue effect, still remains the medication of choice.

SUMMARY AND CONCLUSION

Epilepsy is an abnormal condition in which recurring convulsive seizures result from various influences upon the central nervous system. The symptomatic epilepsies are produced by discoverable disease processes, while the etiology of the idiopathic epilepsies has as yet, not been discovered.

Some fifteen years ago, Sodium Diphenyl Hydantoinate was found to be efficacious in lessening the frequency of convulsive attacks in grand mal epilepsy. The most spectacular tissue reaction associated with the use of this anticonvulsant was a hyperplasia of the gingivae. Beginning as a painless generalized enlargement of the interdental papillae, the lesion later involved the remainder of the gingivae. An initial, stippled granular appearance was followed by a warty, lobulated growth with cleft formations.

The clinical and histopathologic considerations of dilantin gingival hyperplasia occurring in Negro patients at the Veterans Administration Hospital, Tuskegee, Alabama have been discussed.

It would appear that like sex, age, weight or height, racial factors have little influence upon the incidence of dilantin hyperplasia. Nor does race seem to influence the histopathological picture or clinical appearance of the tissues insofar as degree, form and consistency of gingival hyperplasia are concerned.

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Calculus Reduction with a Mucinase Dentifrice

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THE use of a dentifrice containing a "mucinase" type enzyme for the inhibition of calculus formation has been suggested recently by Stewart.[1] In view of the importance of gingival irritation by calculary deposits as an etiological factor in periodontal disease, this approach to the problem is deserving of further consideration. While the problem of calculus deposition can be controlled adequately by routine periodic scaling in the majority of cases, it is well known that there are people who exhibit a prolific rate of calculus formation. It is with these abnormal cases that a special "calculus-inhibiting" dentifrice would find its most useful application.

The term "mucinase" does not describe a single specific enzyme but is a general term describing preparations which have the ability to hydrolyze mucin-like material. Mucin is a glycoprotein and "mucinase" preparations show both proteolytic and amylolytic activity. The relative levels of the two specific types of activity depends on the type of enzyme preparation. If it is assumed that the macroscopic calculary deposits observed clinically develop around minute foci of crystallization (which is reasonable by analogy with other crystallization processes) and that these initial centers are held on the teeth by extremely viscous deposits of mucin-like material, then one might expect that reduction of the quantity or viscosity of the

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mucin-like material would allow more easy removal of these crystallization centers during normal home oral hygiene procedures. If the foci can be continually removed as they form, the chances for continued deposition of insoluble salt to form macroscopic accretions would be materially reduced. Zander[2] has described the various modes of attachment of calculary particles based on microscopic observations. The desired action of the "mucinase" preparation is not concerned with the removal of these well-established deposits, but depends, for its success, on the removal of the foci before any degree of accretion is attained. Therefore this enzymatic approach is not intended to replace thorough prophylactic scaling but is designed to increase the efficiency of home care after adequate clinical treatment. The possible validity of the above assumptions and dependent conclusions were indicated by the successful results originally reported by Stewart.[1]

Because of the non-specific nature of the "mucinase" enzymes, it was felt desirable to test several different types of preparation in an effort to select the most suitable one before initiating large-scale clinical evaluation programs. Further, since the original experiment reported only three cases and had no independent control group, it was of interest to confirm that the observed effects were actually due to the "mucinase" content of the experimental dentifrice. To these ends, the study described below was carried out.

EXPERIMENTAL

Materials. Three experimental powder dentifrices and a control powder dentifrice were used. Powder was used in preference to paste because of the problem of the stability of the enzymes. The powders were distributed in glass bottles labelled only by number and letter code. The three enzyme concentrates were a papain, Prolase 100* and Mylase L-1* and were mixed with a powder dentifrice at a concentration of 3 per cent. The vehicle was a standard formulation used as vehicle for penicillin dentifrice. After packaging, one sample of each group was assayed for enzymatic activity by the laboratories of the Wallerstein Company. The results are shown in Table 1.

TABLE 1
Summary of Materials and Activities

Code	*Enzyme*	*pH*	*Proteolytic Activity*
4902-A	Control	9.3	none
4902-B	Prolase 100	8.6	no loss
4902-C	Mylase L-1	9.2	no loss
4902-D	papain	9.1	20% of calculated

Method. All clinical procedures and observations were carried out by the same investigator (A.A.). At no time during the entire course of the experiment did this investigator know how the three experimental and one control dentifrices had been coded. All records were kept and conclusions drawn using code letters. A group of 11 subjects were selected with the characteristic of more than average calculus formation. In each case a thorough examination was made and the amount and distribution of calculus deposits was noted. Thorough prophylaxis was then performed and the patient was given a supply of one of the dentifrices. Each subject used the assigned dentifrice for six months and was re-examined every month during the study. Each subject used the same dentifrice throughout the entire experiment except those initially assigned to "D" who were reassigned to "C" after three months. All patients were instructed to continue in their regular oral hygiene procedures except for the substitution of the new dentifrice for whatever they had been using previously.

RESULTS

Dentifrice A (Control). Three subjects were assigned to this dentifrice. No im-

*Registered trade-mark of the Wallerstein Company, Inc., 180 Madison Avenue, New York, N. Y.

provement was noted. Redeposition of calculus and some discoloration of the teeth were observed.

Dentifrice B (Prolase 100). Three subjects were assigned to this dentifrice. Reduction in discoloration was noted. Some reduction in the amount of calculus was also observed, and the calculus that had formed was of a softer nature.

Dentifrice C (Mylase L-1). Of the three subjects assigned to this dentifrice, two showed a marked reduction in calculus formation. The small amount of material that had accumulated was of a soft, moist nature. Stain on the surfaces of the teeth was considerably reduced. In the third case the reduction in calculus formation was not as marked but a definite improvement in the situation was apparent. It was suspected that home oral hygiene care was below average standards in this case.

Dentifrice D (Papain). The two subjects using this dentifrice showed no improvement by the end of three months. At that time the subjects were reassigned to "C" and a marked improvement followed. Discoloration of the teeth was reduced and further calculus formation occurred at a reduced rate with a change in consistency to softer material noted in the new deposits.

DISCUSSION AND CONCLUSIONS

Because of the great difficulties encountered in establishing a quantitative method for evaluating the extent of calculus deposition in a mouth, the conclusions here are based completely on clinical observation. The authors feel that a definite reduction in calculus formation can be attained by the use of a "mucinase" dentifrice since a striking difference was observed between the subjects using "A" and "D" and the subjects using "B" and "C." This finding is in agreement with the original report by Stewart.[1] Further, it is interesting to note that with dentifrice "B" or "C," the small amount of new material that was deposited was soft enough to be nonirritating and could be removed by methods less rigorous than the usual scaling. Although it is difficult to be sure on the basis of a small number of patients, the authors feel that these experiments indicate Mylase L-1 ("C") to be the best enzyme preparation of the three tested.

The enzyme concentration of 3 per cent was chosen from the work of Stewart.[1] A question remains as to whether or not this is the most efficient level to use. This can only be resolved by further investigation. It was the purpose of this study only to examine the possibilities of a "preventive" approach to the calculus problem. The possibilities have been found to be quite encouraging and further consideration of the details of "mucinase" therapy is highly recommended.

It is not possible at this time to relate definitely the inhibition of calculus formation with a specific type of enzyme action. Some indications can be obtained from the observation that Mylase was more active in this respect than Prolase and from the fact that the major activity of Mylase is amylolytic while that of Prolase is proteolytic. However these preparations also contain a large number of other enzymes in small, unknown concentrations. This paper, therefore, reports only the finding that a specific preparation will reduce calculus formation and is not intended to call attention to any aspects of the etiology of calculary deposits.

ACKNOWLEDGMENT

The authors wish to express appreciation to the Wallerstein Company for supplying the enzyme concentrates and to the Bristol-Myers Company for the preparation and packaging of the experimental dentifrices.

SUMMARY

1. Three enzyme preparations, Prolase 100, Mylase L-1 and papain, in a powder dentifrice vehicle were tested in comparison to a control dentifrice for ability to prevent or inhibit calculus formation *in vivo*.

2. Under the conditions of the experiment, it was found that Mylase L-1 was extremely effective in reducing stain and preventing calculus formation. Prolase 100 was effective but not to the same degree as Mylase L-1.

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Tooth Mobility (II)*

The Role of Interdental Contact Points and of Activation on Tooth Mobility

by HANS R. MÜHLEMANN, M.D., D.D.S.,** *Minneapolis, Minn.*

THERE are several factors which may lead to errors in the interpretation of tooth mobility*** measurements. The fact was stressed[1,2] that the measurements should always be performed under standardized conditions. Observing these precautions the reliability of the method was shown.[1,2] Since we deal with living structures on which we use the measuring methods, there are other factors inherent to the teeth and their surrounding structures which must also be taken into consideration when evaluating TM values. It is the purpose of this communication to show the role which two such factors have in the interpretation of tooth mobility measurements.

MATERIAL AND METHODS

Seven Rhesus monkeys were used for the study. The methods employed have been described.[2] The results and conclusions are based on 460 measurements of 30 teeth.

RESULTS

1. *The effect of interdental contact points on tooth mobility.* The influence of contact points on tooth mobility is illustrated in Figure 1. TM measurements were made with forces of 100 and 500 grams on two upper central incisors on an adult monkey. These teeth were well aligned. After these measurements, the mesial and distal tight contact points of the incisors were removed with carborundum discs. The measurements were then repeated. It was found that removal of contact points did not affect initial TM but that the secondary TM (T_{500}) was increased in both teeth. This effect was confirmed on many other cases. In all of them there was an augmen-

*This is the second of a series of reports about tooth mobility.

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***The abbreviation TM designates tooth mobility.

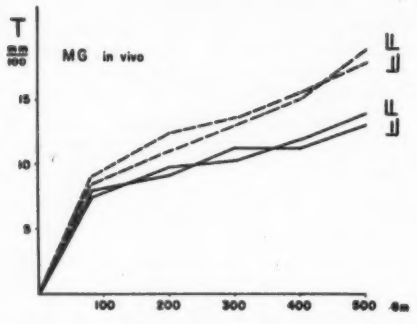

Fig. 1. The effect of interdental contact points on tooth mobility. Measurements before (———) and after (— — — —) removal of mesial and distal contact points.

tation in the slope gradient of the secondary TM.

The mechanism of the contact points' effect upon tooth mobility can also be demonstrated with another procedure (Fig. 2). In Figure 2 the upper TM curve was obtained by the usual technic on the left upper central incisor (|1). The lower curve demonstrates the movement of the *right* central incisor (1|) when the forcemeter was applied not on this tooth but on the crown of its neighbor, the upper *left* central incisor. The movement of 1| started with low forces (100 grams) and remained at the same level when higher forces were applied to the other central.

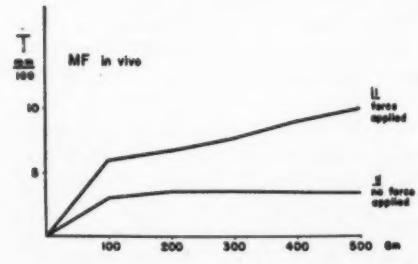

Fig. 2. Effect of interdental contact points on TM. The upper TM curve was obtained by the usual technic. The lower curve shows the crown excursions of the right upper central incisor when the forces are applied on the left upper central incisor.

2. *The effect of multiple continuously repeated measurements on tooth mobility.* The measuring method itself can also change the degree of TM. If measurements are continuously repeated on the same tooth, mobility increases (Fig. 3). The first series of measurements on the upper right incisor of an adult monkey is shown in a heavy solid line in Figure 3 (rest TM). Measurements were continued for two hours with short lasting forces from 100 to 1000 grams. The curve for the second series of measurements was found to be above the curve of rest TM. The third, fourth, and all the following curves fell

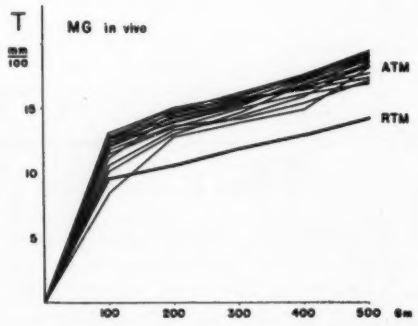

Fig. 3. Activated tooth mobility. RTM—Rest TM obtained by the first measurements. ATM—Range of activated TM obtained by continuously repeated measurements.

within the same range, showing no further increase of TM (saturation level). The range into which these latter curves fell is called the range of *activated tooth mobility* (ATM) in order to distinguish it from the first series of measurements which will be called *rest tooth mobility* (RTM). The increase of TM by activation is rather small ($T_{500} < 5/100$ mm in an adult dentition). It is mainly due to an increase of *initial TM*, the slope gradients of activated and resting secondary TM being similar (Fig. 3). This can also be seen in Figure 4 where TM augmentations due to activation are demonstrated on four upper

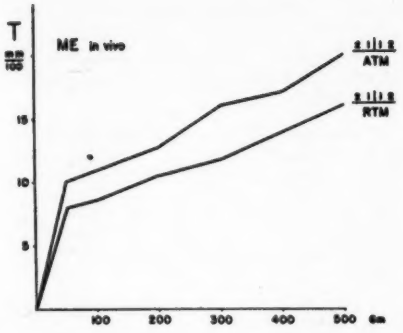

Fig. 4. Transformation of rest tooth mobility (RTM) into activated TM (ATM) by activation. (Averaged TM curves from four incisors.)

incisors. The average RTM of these teeth was combined into one curve, as well as the average ATM.

The recovery of ATM and its return to RTM was also studied. It was found that the recovery is quite rapid. Figure 5 shows a rest TM curve (curve 1) of an upper incisor and the increase of TM by prolonged and uninterrupted activation with forces up to 1000 grams (curve 2). Curve 3 was obtained after a rest period of 25 minutes. TM returned to almost its initial resting values. TM was again activated by holding a force of 800 grams against the tooth for 60 seconds. Curve 4

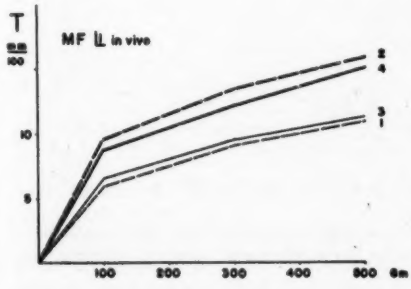

Fig. 5. Recovery of activated tooth mobility. 1—Rest tooth mobility. 2—Curve from the activated TM range. 3—TM curve obtained after a rest period of 25 minutes. 4—TM curve obtained after reactivation.

was obtained immediately after removal of the 800 grams.

DISCUSSION

The results have shown that tooth mobility may be influenced by the presence of interdental contact points and by activation. Contact points can produce lower values of *secondary TM*. These differences are small ($T_{500} < 5/100$ mm) and, using the present measuring method, they are only of practical importance if contact points are removed or altered between measurements that one wishes to compare.

The contact point effect is, however, one of the most serious problems when not static but instead vibrating forces are used for the assessment of mobility.[3,4] It is difficult to agree with Manly, et al.[4] when they write, "The problem of measuring mobility is considerably simplified when the force is applied as a vibration rather than a static force." Their clinical results were inconsistent. This was most likely due to interdental contact and friction which change very irregularly the vibration pattern of teeth. This is much more serious than the effect of contact points upon TM when static forces are used for its evaluation.

The transformation of rest tooth mobility into activated tooth mobility is associated with an increase of the initial TM. The slope gradients of secondary RTM and secondary ATM do not change significantly. Activation of TM does not occur if only a few measurements are performed on the same tooth during routine measurements. The phenomenon of activation occurs under heavy and continuously repeated forces.

Activation can also be observed under non-experimental conditions. It was shown[1] that uninterrupted, 15-minute periods of masticating hard food (hazel nuts) increased the "maximal TM"[2] of upper incisors about 5/100 mm. Therefore it is advisable to take into consideration the functional status of a dentition if very small TM differences are to be compared. It is recommended to avoid such measure-

ments shortly after periods of mastication and to check chewing habits prior to measuring.

CONCLUSIONS AND SUMMARY

It was shown on Rhesus monkey teeth that interdental contact points can influence the amount of labio-lingual crown excursions. Measurements continuously repeated for several minutes on the same teeth lead to a slight but consistent increase of initial tooth mobility. Recovery from this mobility increase may occur within half an hour.

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Tooth Mobility (III)*

The Mechanism of Tooth Mobility**

by HANS R. MÜHLEMANN, M.D., D.D.S.,*** AND HELMUT A. ZANDER, M.S., D.D.S.,

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IN two previous reports[1, 2] the instantaneous crown excursions associated with the application of lingual or labial forces to teeth were studied. The magnitude of these excursions did not increase in a linear proportion when forces increasing in magnitude were applied, thereby allowing a differentiation between initial and a secondary tooth mobility (TM). It was obvious that initial and secondary TM could be detected only because of differences in tissue resistance. The present study was undertaken in order to visualize the intra-alveolar displacement of the roots during TM measurements and to compare the positions of the roots with the registered crown excursions. What is the intra-alveolar position of the roots during the phases of initial and secondary TM? Where is the rotation center of the tooth when linguo-labial forces of different magnitudes produce the crown excursions? Is TM due only to an intra-alveolar displacement of roots or are elastic deformation and distortion of periodontal tissues also involved?

MATERIAL AND METHODS

1. *Determination of the rotation center of teeth.* Tooth mobility was measured on monkey teeth at two different places of the crowns: near the incisal edge (I) and near the cervical region (C) (Fig. 1). Knowing the crown excursion at I (= i) and at C (= c) for a given force, and knowing the distance between I and C (= d) for a given force, the approximate location of the rotation center (RC) could be calculated ($x = \frac{c \cdot d}{i - c}$)† (Fig. 1). After sacrificing the animals and obtaining histological sections from the measured teeth, photomicro-

*This is the third of a series of reports about tooth mobility.

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†This formula represents only the principle underlying the determination of rotation centers. It needs many complicated corrections due to the curvature of the crown surface.[3]

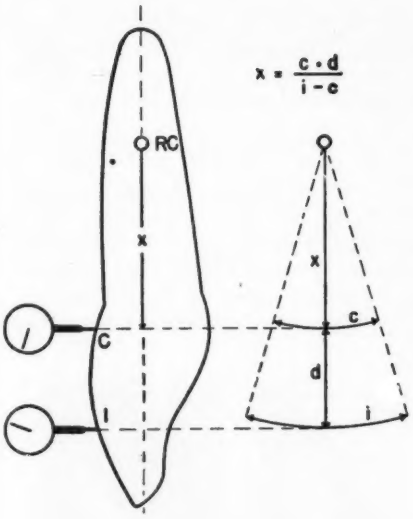


Fig. 1. Principle of the determination of the instantaneous rotation center of teeth with TM measurements. i—Total crown excursion measured incisally at I. c—Total crown excursion measured cervically at C. d—Distance between I and C. RC—Rotation center determined by $\times = \frac{c \cdot d}{i - c}$

graphs were made from these sections. Prints from these photomicrographs were enlarged 10 to 20 times. The teeth on the enlarged prints were cut out and used for determinations of the rotation center of the teeth measured.

2. *The intra-alveolar displacement of roots during tooth mobility measurements.* A visualization of the in vivo intra-alveolar position of the root when different forces are applied to the crowns, was accomplished in the following way: Well fitting palatal silver plates were constructed for three Rhesus monkeys. Into these appliances screws were inserted and directed towards the lingual surface of upper incisors, or premolars and/or molars (Fig. 2). By advancing a screw the crown could be tipped in a labio-buccal direction. In order to push this crown by the screw with a known force, the dial indicator was applied on the labial side of this tooth. If the intra-alveolar position of the root was to be studied, e.g. under a force of 100 grams, such a force first was applied to the crown from a lingual direction with a forcemeter and the position of the hand on the dial indicator was recorded. Next the screw was advanced until the crown moved to the position obtained previously with the forcemeter. With other teeth the same procedure was followed using 300 grams. In order to hold the crown firmly in the tipped position, self-curing acrylics were inserted over and between the silver plate and the lingual surface of the crown. Within five minutes after positioning and holding the tooth the monkeys were sacrificed; the whole upper jaw with the appliance in situ was separated from the skull and immersed in Zenker-formol. During fixation and at the time of the removal of the appliance, care was taken to check a possible rebouncing of the crowns or receding of the appliance. Afterwards, the teeth in situ were sectioned labio-lingually and stained with hematoxylin and eosin. The sections showing the narrowest periodontal membrane on the labial and lingual sides were photographed and enlarged prints were made. Knowing the amount of crown excursion in vivo and

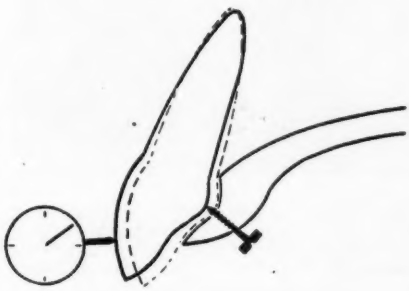


Fig. 2. Labial crown excursion produced by screw inserted in palatal plate. The dial indicator measures by means of a previously established TM curve the force applied.

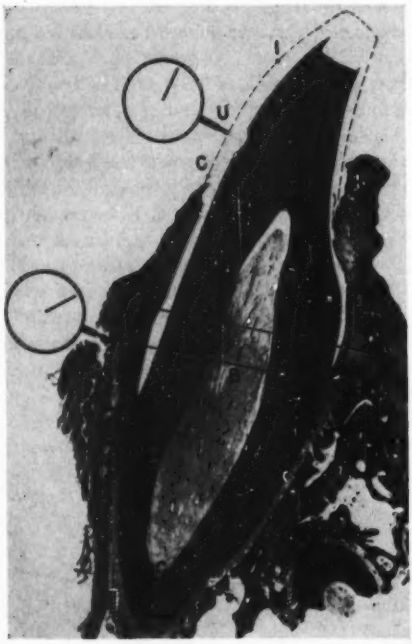


Fig. 3. Measurement of tooth mobility at U and of bone distortion at B-level (B). I and C were used for the determination of the rotation center, RC. (Monkey MC | 3).

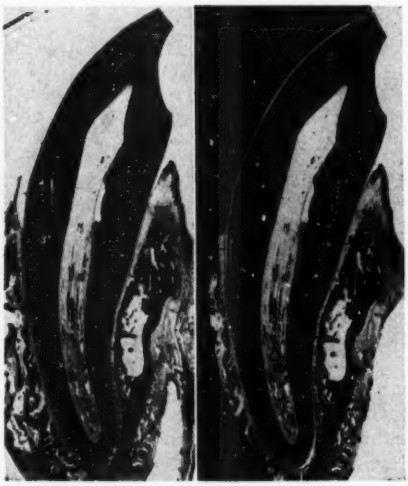
knowing the determined location of the rotation center, it was possible to reposition the cut-out teeth on the prints from their labial position back to their rest position. Corrections indicated by geometrical reasons were made on the prints when necessary.

3. *Elastic deformation of the alveolar bone.* TM measurements first were made in the usual manner, with the dial indicator applied to the crown at "U" (Fig. 3). After these measurements which allowed the construction of TM curves the contact point of the dial indicator was placed in direct contact with the labio-marginal alveolar bone at the "B-level"* through an incision of the attached gingiva. The same crown was moved again with known forces labially and lingually, and the deformation of the supporting labio-marginal alveolar bone registered on the dial indicator.

*The "B-level" was defined as a level located 1 mm. apically from the labio- or lingua-marginal alveolar crest.[1]

RESULTS

1. *Determination of the rotation center of teeth.* The crown excursions when measured in the region of the incisal edge were always greater than when registered in the cervical region of the same crowns, suggesting a tooth movement around an instantaneous rotation center situated apically from the cervical region. The location of this rotation center varied between different teeth and when different forces were used. Rotation centers for three teeth are shown in Table I. The rotation centers changed position when different forces were applied to the crowns, when the measurements were performed with and without contact points, in vivo and post mortem. There were also differences in the position of rotation centers in activated



A B

Fig. 4. A—Intra-alveolar position of root held labially with 100 grams. (MC | 1). B—Reconstructed rest position of tooth in Fig. 4-A. (RC—rotation center; B—B-level).

TABLE I

Localization of Rotation Center of Monkey Teeth Determined by Tooth Mobility Measurments

Monkey and Tooth	*Force Applied Gms.*	*Rotation Center. Distance from Cervical Region Towards Apex.**				
		In Vivo			*Post Mortem*	
		With Contact Points	*Without Contact Points*		*Without Contact Points*	
			Non-Activated	*Activated*	*Non-Activated*	*Activated*
MF $\underline{1}$	100	6.5	6.3	3.8	4.6	3.2
	200				6.0	5.3
	300	7.4	6.3	4.1	6.6	6.3
	400				18.0	6.3
	500	8.1	6.0	4.8	15.3	6.2
MF$\underline{1}$	100			4.7		5.8
	200					8.3
	300			4.1		9.3
	400					9.0
	500			5.5		8.9
MC$\underline{3}$	100		12.1			
	200		10.8			
	300		12.9			

*Distance in millimeters (see Fig. 1).

and non-activated teeth.[2]

2. *The intra-alveolar displacement of roots during TM measurements.* The described method allowed a non-relapsing holding of the crowns. The enlarged photographic prints showed the positions taken by the roots when the crowns were held labially during TM measurements with 100 or 300 grams. In Figures 4-A and 5-A the intra-alveolar positions of two upper incisor roots of the same monkey (MC) when held with 100 grams and 300 grams, respectively, are illustrated. It can be seen in both sections that the width of the periodontal membrane on the labio-marginal side (B-level) is less than that on the linguo-marginal side. In Figure 4-A the crown was positioned with 100 grams; the width of the labio-marginal periodontal space was 76 per cent of the width of the linguo-marginal periodontal membrane. In Figure 5-A, where 300 grams were applied, it was 41 per cent. Repositioning of the two incisors was performed using the data from the TM measurements. After this repositioning it was found that the width of the labial periodontal membrane was, at B-level, 90 per cent of the linguo-marginal periodontal membrane width (Fig. 4-B) and 62 per cent (Fig. 5-B), respectively. Knowing the widths of the labial and lingual periodontal membrane when the teeth were held labially and knowing their repositioned rest position, the percentage changes of membrane width on the pressure and the tension side (B-level) when applying 100 grams and 300 grams, respectively, were calculated (Table II).

An increase or decrease of membrane width was also evaluated by using specimens showing roots in rest position and from which TM values were available. Positioning these teeth labially and lingually on the enlarged prints as if forces of 100 grams had been used gave further indication of the amount of periodontal width changes at B-level. The data are summarized in Table III.

TABLE II

Decrease or Increase of the Labio- or Lingue-Marginal Periodontal Membrane Width During Initial and Secondary Tooth Mobility

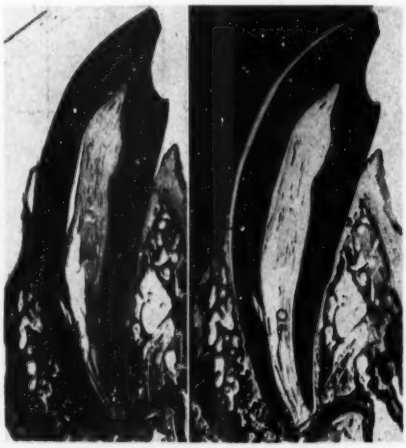
	Monkey and Tooth	% of Decrease in Width on Labial Pressure Side*	% of Increase in Width on Lingual Tension Side*
Initial TM (100 gms)	MC $\underline{1}$	8.5	12.2
Secondary TM (300 gms)	MC $\underline{1}$	24.6	12.4

*At B-level.

The initial TM was on the upper left central, and the secondary TM on the upper right central, instead of as shown.

An incidental observation made on the histological sections of teeth held labially with 100 or 300 grams was vascular stasis (Figs. 6 and 7, which are enlargements of Figs. 4 and 5). It can be seen in those specimens that the circulatory disturbance is present in the periodontal membrane on the pressure side in the apical region and also in the alveolar bone. Figure 8 is from the same area of a tooth to which no forces had been applied. Note the absence of circulatory disturbance. Similar circulatory disturbances were observed on multirooted teeth tipped and held with 100 grams (Fig. 9-A). Note the particular distribution of stasis on the three molar roots and the absence of hyperemia on the roots of a control tooth (Fig. 9-B).

3. *Elastic distortion of the alveolar bone.* In *young* monkeys a distortion of the alveolar bone during TM measurements started when forces greater than 100 grams were applied. In Figure 10 the upper curve represents the activated total TM of an erupting upper cuspid (MC $\underline{|3}$) and the lower curve shows the amount of elastic distortion of the marginal alveolar *bone* that occurred during the *tooth* mobility measurements used for the construction of the upper TM curve. Comparing the TM curve with the curve of the elastic bone deformation it may seem that the amount of the bone deformation is relatively small. However, it has to be remembered that the crown excursions at the point where *tooth* mobility was measured were much greater than the tooth excursions at the B-level where the bone deformation was registered.

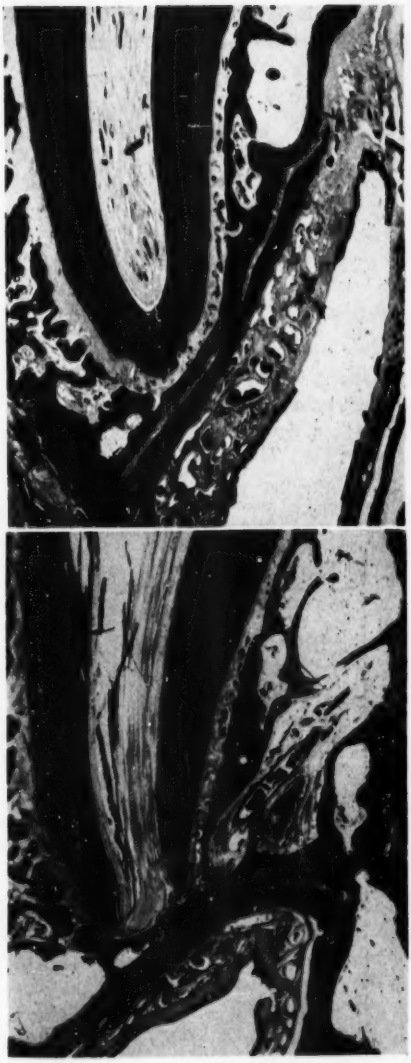


A B

Fig. 5. A—Intra-alveolar position of root held labially with 300 grams (MC $\underline{1|}$). B—Reconstructed rest position of tooth in Fig. 5-A. (RC—rotation center; B—B-level).

An approximate idea of the degree of the tooth displacement at the level of bone deformation (Fig. 3) was obtained by determining first the rotation centers (Table I, monkey MC $\underline{|3}$). With the knowledge of the location of these centers the displacement of the tooth at the bone level was calculated. This calculated "root"* mobility curve is shown in Figure 11. The data presented in Figures 3 and 11 provide the following information:

*The term of "root" is used to avoid confusion. It can be seen in Figure 3 that it was a part of the anatomical crown which was distorting the alveolar bone. (Tooth in the initial stage of eruption!)



Figs. 6 & 7. Vascular stasis in apical pressure zone of periodontal membrane and adjacent bone of teeth held labially during five minutes with 100 grams, and 300 grams. See control tooth in Fig. 8.

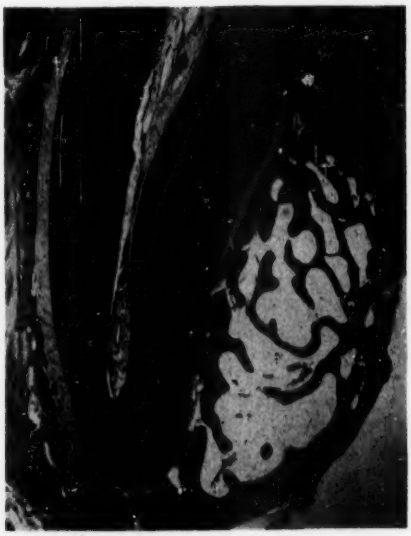


Fig. 8. No signs of circulatory disturbance in control tooth.

1. Forces of 100 grams produced (Fig. 3) a total crown excursion of T = 39/100 mm; 300 grams produced an excursion of T = 60/100 mm, at U.

2. From these data the total "root" displacement at the bone level (which coincided with B-level) was calculated as 11/100 mm for the 100-gram force and as 17/100 mm for 300 grams.

3. Only the total "root" displacement with 300 grams was associated with a distortion of the marginal alveolar bone. The bone movement during the total tooth excursion was 7/100 mm.

4. Taking into account the membrane widths at rest position (0.486 mm labio-marginally, 0.324 linguo-marginally, at B-level) and the bone distortion, the changes of membrane width on the *labial* pressure side and on the *lingual* tension side were found as listed in Table IV.

DISCUSSION

The results have shown that physiological tooth mobility is due to intra-alveolar displacement of the root and bone

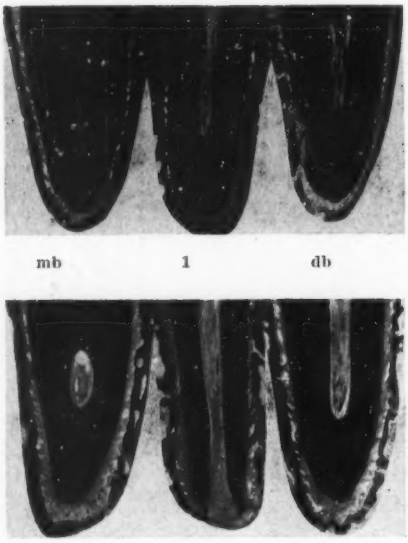


Fig. 9. A—Mesiobuccal (mb), lingual (l) and distobuccal (db) roots of upper molar (ME $\underline{6}$) held buccally with 100 grams with zones of stasis. B—Control tooth with no forces applied MB $\underline{6}$).

distortion. The magnitude of the force applied labially or lingually to the crown decides whether the crown excursion is associated with root displacement only or with root displacement and bone distortion.

In order to understand the mechanism of tooth mobility the occurrences in the alveolar socket during the initial phase of TM were studied first. It had been shown previously[1] in a study of TM curves that the resistance offered by the tooth supporting tissues against moving or tipping of the crown is very low in the intial phase of TM. Within the range of initial TM (produced by forces up to 100 grams) the crown excursions are relatively large, and so must be the intra-alveolar root displacements. This initial intra-alveolar movement of roots leads to changes of the width of the periodontal membrane. For example, a histologic section of a tooth that was positioned labially (in vivo) with a force of 100 grams (Fig. 4-A) showed at the labio-marginal B-level a reduction of the membrane width by 8.5 per cent and an increase in width on the linguo-marginal tension side of 12.2 per cent (Table II).

The study of specimens in which the teeth were positioned with forces higher than 100 grams showed the important fact that the *linguo*-marginal extension side remains constant in its width despite the increase of the tipping force (Table II). On the other hand, the *labio*-marginal periodontal membrane width was reduced further.

These findings mean that the initial displacement of a root from its rest position towards a new position is obtained with relative ease. The same is true initially for

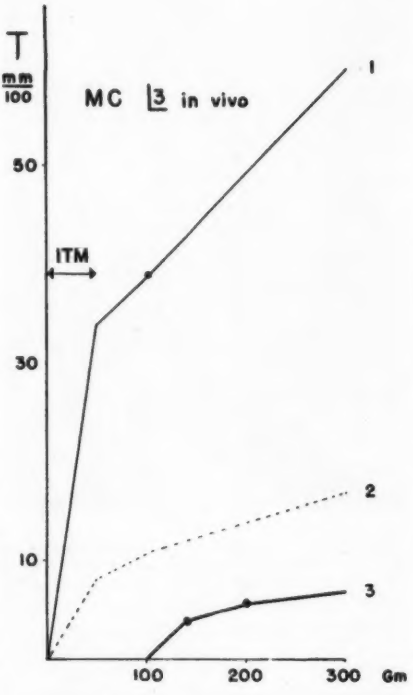


Fig. 10. 1—Activated tooth mobility at U (Fig. 3). 2—Theoretical "root" mobility curve. 3—Distortion of alveolar bone during TM measurements. ITM—Range of initial TM.

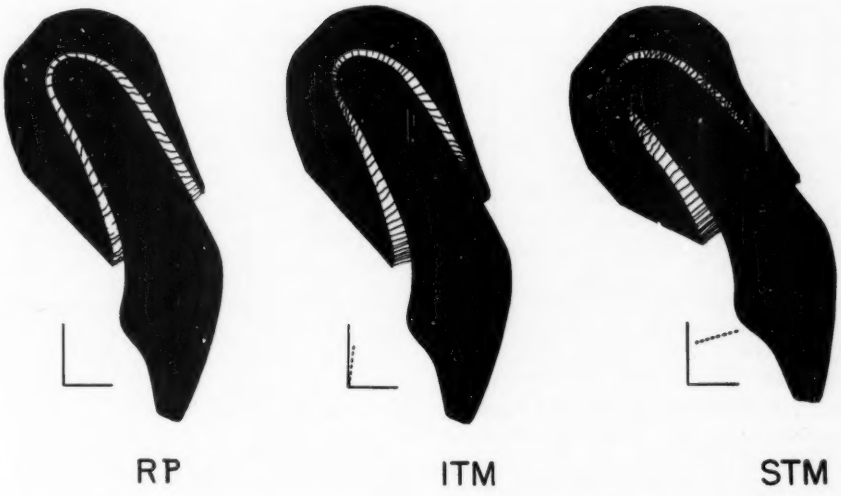


Fig. 11. Mechanism of tooth mobility. RP—Intra-alveolar rest position of root. ITM—Initial tooth mobility with intra-alveolar displacement of root and reorientation of periodontal membrane fibers towards functional readiness on tension side. Initial phase of TM curve. STM—Secondary tooth mobility with further intra-alveolar root displacement and simultaneous tissue compression and distortion on pressure side. Secondary phase of TM curve.

the associated changes in membrane width on the pressure and tension sides. There are good reasons to assume that the initial displacement of the root (initial TM) corresponds to a reorientation of the periodontal membrane fibers into a position of functional readiness towards tensile strength: It was previously[1] shown that initial TM is highly dependent on the structure of the periodontal membrane. Initial TM and the initial intra-alveolar root displacement are small in membranes where the fibers already show a distinct orientation in rest position. Initial TM is much greater in the presence of loosely and poorly structured membranes. This morphological difference is also the cause of different membrane width changes which were found in this study (Table III). On the tension side of teeth in heavy function and with well structured periodontal membranes, the orientation of the periodontal fibers to functional readiness was associated with a membrane width increase ranging between 3.3 and 4.5 per cent only. In an erupting cuspid with poorly structured membrane (Fig. 3 and Table III) the increase in width reached 17.7 per cent on the tension side.

The fact that the linguo-marginal membrane width was found to remain constant after the initial phase* of TM provides an explanation for the second phase of TM. The passage from initial to secondary TM occurs because the membrane width of the tension side cannot be further increased with forces higher than 100 grams. The fiber bundles resist any further root displacement. The extensibility of collagenous fibers is practically negligible. This new resistance is illustrated in TM curves by different slope gradients.

The physical factor involved in the second phase of TM was found to be distortion of the whole periodontium. The *labial* bone distortion, for example, in the case illustrated in Figure 10 where 300 grams were applied to the lingual surface of the crown, was 0.035 mm. The result of com-

*Labial excursion.

TABLE III

Decrease or Increase of the Labio- or Linguo-Marginal Periodontal Membrane Width During Initial Tooth Mobility

Monkey and Tooth	*Stage of Eruption*	*% of Decrease in Width on Labial Pressure Side**	*% of Increase in Width on Lingual Tension Side**
MC$\underline{3}$	Initial	14.8	17.7
MC$\underline{1}$	Terminal	8.5	12.2
MA$\underline{1}$	Terminal	8.3	9.4
MF$\underline{1}$	Completed	3.0	4.5
MF $\underline{1}$	Completed	2.1	4.0
ME $\underline{1}$	Completed	3.2	3.4
ME $\underline{2}$	Completed	3.5	3.3

* At B-level.

bined measurements of TM and bone distortion showed again that the periodontal membrane width does not change on the tension side after the initial displacement of the root (initial TM) despite the use of forces strong enough to distort the alveolar bone (Table IV). This was a valuable confirmation of the same finding (Table II) which was obtained by a different method.

In view of these facts one could think that the mechanism of tooth mobility consists first of an initial displacement of the root and then of bone distortion. However, such an explanation of TM is not sufficient without consideration of a third occurrence which is apparent from Figure 10: If, for example, a 120-gram force was applied to the crown, the total tooth displacement was 11.5/100 mm at B-level. This displacement was due:

1. to root orientation during initial TM and amounted to 8/100 mm.

2. to bone distortion of 2/100 mm.

The amount of displacement of (1) and (2) added together makes 10/100 mm. Since the root displacement was 11.5/100 mm there is 1.5/100 mm not accounted for. This difference is due to *tissue compression*, which is the third factor in the mechanism of tooth mobility.

Another example of membrane compression (MC $\underline{1}$ was given in Figure 5 and Table II. It was shown that the periodontal membrane on the *pressure* side was reduced further in width by forces greater than 100 grams. Soft tissue compression varies

TABLE IV

Distortion of Marginal Alveolar Bone, Changes of Periodontal Membrane Width During Initial and Secondary Tooth Mobility. (Erupting Tooth, MC$\underline{3}$)

	*% of Decrease in Width on Labial Pressure Side**	*% of Increase in Width on Lingual Tension Side**	*Amount of Labial Alveolar Bone Distortion in mm.*
Initial TM T=0.39 mm L=0.195 mm	11.2	15.2	0
Secondary TM T=0.60 mm L=0.30 mm	11.3	15.4	0.035

*At B-level.

considerably. It was greatest when the alveolar bone was of low "elasticity" (functioning teeth); it was less pronounced in erupting teeth of young monkeys. Differences in the thickness of the labial or lingual alveolar bone plate seem also to affect the tissue compression.

All three fundamental factors involved in the mechanism of tooth mobility are represented schematically in Figure 11.

An incidental finding was the appearance of vascular stasis in the periodontal membrane and in regions adjacent to it (Figs. 6-9). The hyperemic zones were only located in the apical pressures areas. They were produced in a few minutes since the animals were sacrificed three to five minutes after positioning the teeth. The local circulatory disturbance was not dependent on the size of the forces used. It was already pronounced when forces not greater than 100 grams held the crowns in tipped position. From the topographical distribution of the areas of pressure and stasis in *multirooted* teeth (Fig. 9) it should be concluded that the intra-alveolar displacement of the roots is very complicated. The distribution of the zones of pressure did not always follow a pattern that would correspond to a simple linguo-labial tipping of the crowns.

The determination of the rotation centers of teeth was valuable and necessary for exploring the intra-alveolar displacement of the roots during TM measurements. These "constructed" rotation centers cannot be compared with the "physiologic" rotation center described by Klein[5] or the "orthodontic" rotation center described by Ketcham[6] and Oppenheim[7] and many others.

CONCLUSIONS AND SUMMARY

The intra-alveolar displacement of roots when different forces are applied to the crowns of monkey teeth was studied with two different methods. TM data were compared with histological sections of the measured teeth. It was found that *initial tooth mobility* is due to an intra-alveolar orientation of the periodontal membrane fiber bundles. Not more than 50 to 100 grams are necessary to prepare the membrane fibers for functional readiness. If forces higher than 100 grams are applied to the crowns, the fiber bundles on the tension side resist any further root displacement. They prevent a further increase of the periodontal membrane width on the tension side. This is the cause for the passage of initial TM into *secondary tooth mobility* and for the characteristic double-sloped TM curves. Increasing the crown excursions with forces higher than 100 grams (range of secondary TM) leads to distortion and compression of the whole periodontium. This distortion is reversible under physiological conditions and more pronounced in young monkeys with high tissue "elasticity."

Changes in periodontal membrane width during the first phase of initial tooth mobility in well functioning teeth of adult monkeys are three to four times smaller than in erupting teeth with poorly structured membranes. Soft tissue compression during the phase of secondary tooth mobility is greater in erupted than in erupting teeth.

The distinction between initial and secondary tooth mobility allows a separate appraisal of the functional structure of the periodontal membrane and of the elasticity of the periodontium as a whole.

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(Continued on page 153)

The Dentin: Its Physical Characteristics During Curettage
II. Statistical Findings

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IN the preceding article[1] we disclosed the presence of a previously unrecorded change in the physical character of the dentin in the cervical area of human teeth and described the gross characteristics of the lesion. Here we present the statistical findings.

It will be recalled that 500 extracted teeth were curetted with a sharp instrument, as is the practice in curettage of the root surface during periodontal treatment, and that measurable amounts of the enamel in the cervical area were usually removed[2]; that the cementum was always removed in its entirety[3,4]; and that a definite characteristic pattern formed as the scraping instrument penetrated deeper and deeper into the dentin, at and adjacent to the cemento-enamel junction.[1] The pattern was that of a wedge shape abrasion, which was not limited to the buccal surfaces, but found on all surfaces. Neither were these grooves of equal depth all around the circumference of all teeth, some surfaces being much more seriously afflicted than others. Nor was it found at all ages, as witness the following.

THE DEPTH OF THE PENETRATIONS

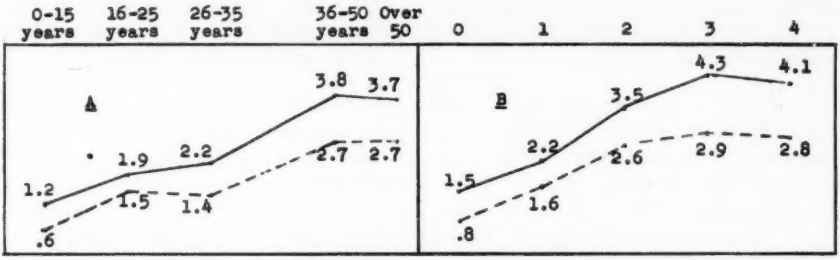
Of first importance in analyzing our findings was to ascertain whether or not the lesion was to be found equally on all teeth at all ages, and, if so, whether or not there was a definite relation to advancing years. It was also important to determine the same in relation to the depth of the pathologic crevice as represented by the clinging attached fibers of the periodontal ligament. We assumed that increasing crevice depth indicates a corresponding increase in the severity of the periodontal involvement before the extraction of the tooth.

Our determinations were made by measuring the greatest thickness of the root at the cemento-enamel junction, through the bucco-lingual diameter and through the mesio-distal diameter, before curettage and again after curettage. The difference between the two would thus represent the overall loss of tooth structure during curettage. Pilot studies had shown us that the cemento-enamel junction was always the site of the deepest penetrations.

Graph 1, A and B represent our findings —the average losses in diameter when classified according to age (*A*) and according to the depth of the pathologic crevice (*B*).

Before sixteen there was no pathologic crevice as represented by the clinging attached fibers of the periodontal ligament,[5] yet we find an overall loss of tooth structure at the cemento-enamel junction of 1.2 mm bucco-lingually and .6 mm mesio-distally. As mentioned in our studies of the cemento-enamel junction,[3,4] this is much greater than the estimated thickness of both the cementum and the granular layer of Tomes in this area. From sixteen to twenty-five, as detachment of the periodontal ligament has exposed the root immediately adjacent to the cemento-enamel junction,[5] there was a corresponding increase in the depth of the penetrations. Then, just as we found in our studies of the location of the bottom of the pathologic crevice,[5] the cemento-enamel junction,[3] and the cementum,[4] there was only a slight increase between twenty-six and thirty-five, as if the process responsible for the deteriorations had slowed or almost ceased. After thirty-five, the penetrations increased in depth rather rapidly, and then again seemed to slow after fifty. That this latter is not true will be demonstrated in a later paragraph.

When the teeth are reclassified according to the depth of the pathologic crevice, regardless of age (*Graph 1 B*), the results are so similar to the age grouping in *Graph 1 A* that separate discussion is not indi-



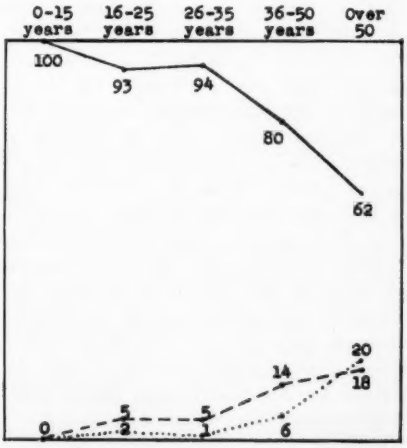
Graph 1. The Average Loss in Diameter in millimeters. 1 cm = 1 mm. A—When classified into Age Groups. B—When classified into groups according to the depth of the pathologic crevice; 0 = attached fibers of periodontal ligament at the cemento-enamel junction; 1 = attached fibers apical to the cemento-enamel junction, but less than ¼ the distance to the apex; 2 = attached fibers ¼ or more, but less than ½ cemento-enamel junction to apex; 3 = ½ or over, but less than ¾ cemento-enamel junction to apex; 4 = ¾ or over cemento-enamel junction to apex.————bucco-lingual; — — — —mesio-distal.

cated, except that the general paralleling of the increasing penetrations with age and with increasing crevice depth seems to indicate a general trend: *Increasing age = increasing crevice depth = increasing depth of penetration.*

Thus we found that the lesion is a progressive one, increasing from a slight deterioration in childhood, to an alarming loss in later years—a loss great enough that some teeth were weakened to the point of stopping curettage before the smooth hard surface was reached, to avoid fracture, and on many to the point that actual fracture occurred.

A depth of penetration of over 4 mm bucco-lingually and almost 3 mm mesio-distally (*Graph 1 B, 3 and 4*) graphically illustrates the surprising depth of these softenings of the tooth structure in old age and in advanced periodontal lesions, particularly as these are average losses, representing an even greater loss on some. It thus seemed essential to determine the incidence of these fractures and near-fractures when subdivided into age groups.

Graph 2 illustrates these findings. Before sixteen, we were able to obtain the hard, smooth surface we deemed desirable in 100% of the teeth, but by twenty-five and from there to thirty-five there were 6-7% of teeth on which we either stopped our scraping to avoid fracture, or actually fractured the tooth before this smooth hardness was attained. After thirty-six, there was a sharp increase in the number of teeth on which it was impossible to obtain our objective, and after fifty, 38% of the teeth could not be planed to smoothness without fracture or near-fracture. Notice that again, the plateau between twenty-six and thirty-five years would seem to indicate a



Graph 2. Ability to Obtain a Hard, Smooth Surface (per cent of teeth). ———— Obtained; — — — — Stopped to Avoid Fracture; ..... Fractured.

slowing-up or a cessation of the deteriorating process, just as it did in the depth of the penetration in *Graph 1*.

In *Graph 1*, as mentioned before, one would get the impression that there was no increase in the depth of the softened tooth substance after fifty years (*Graph 1 A*, 36-50 years and over 50; *B*, 3 and 4). That this is not true is shown in *Graph 2*, where the incidence of fractures and near-fractures increases just as rapidly after fifty as in the period before, a situation which surely indicates that the softening process did continue, and markedly so. The explanation of this disparity in *Graph 1* seems to be that a corresponding surface wasting during this age period, evidenced by previous studies of the surface of the cementum,[3, 4] probably made the before-curettage measurements enough smaller that the after-curettage determinations failed to show that the penetrations were increasingly deep, though we were definitely closer to the pulp than in the thirty-six to fifty grouping.

VARIATIONS IN THE DEPTH OF THE PENETRATIONS

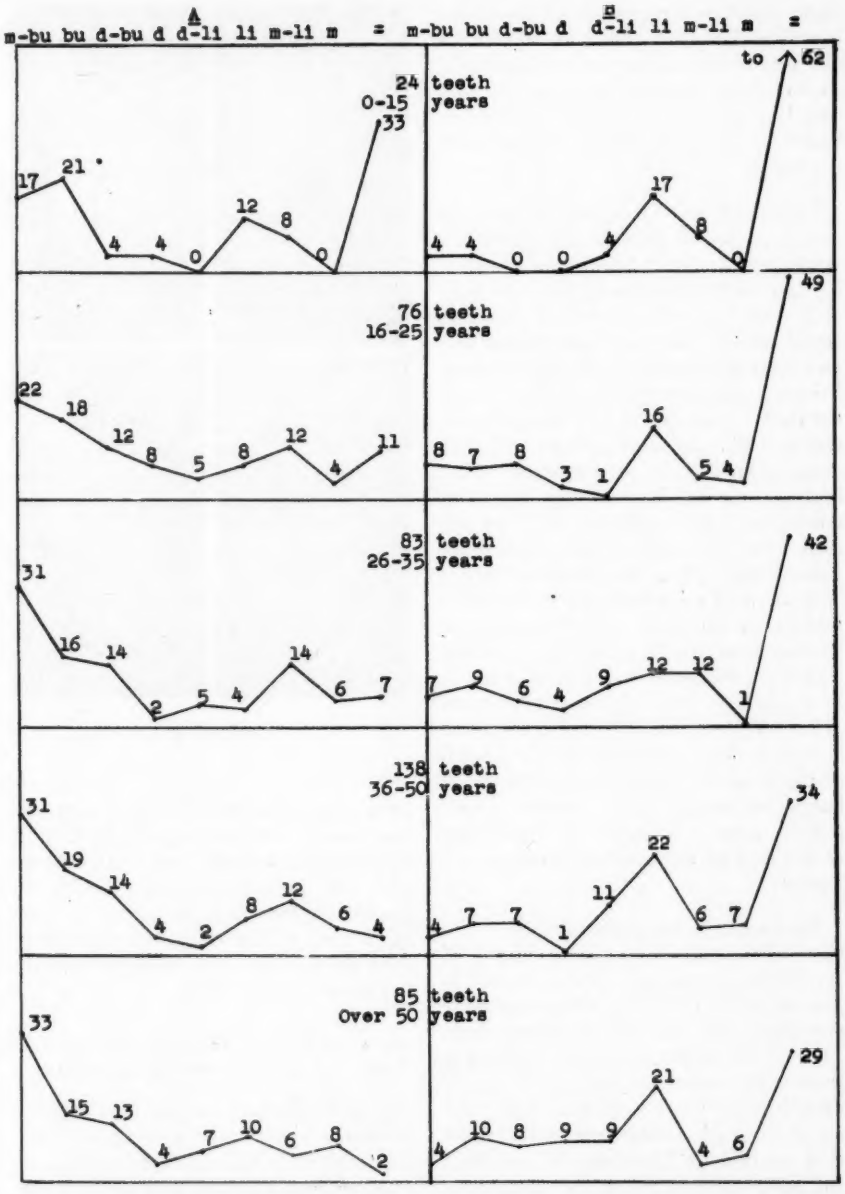
Were these penetrations always of equal depth completely around the tooth, statistical evaluation would be simple, but they are not. Referring to *Graph 1*, it becomes obvious that the softenings are always deeper in the bucco-lingual diameter than in the mesio-distal; i. e. the damage is greater on either/or the buccal and lingual or both, than it is on the mesial or distal or both, but this does not necessarily infer that the losses are of equal depth. Rather, they are more commonly of unequal depth. In part I of this study[1] we described this variability and pointed out the existence of markedly deep areas on most teeth, characteristics so manifest that they could be defined as "Areas of Deepest Penetration" and "Areas of Lesser Penetration."

Again, were these areas located directly on the buccal and/or lingual, mesial and distal, our study would be simplified, but they are not. They are found on all surfaces, varying from patient to patient, tooth to tooth, and year to year.

In *Graph 3 A* we depict our findings when the tooth surface location of the Area of Deepest Penetration is charted according to Age Groups. There were only a few teeth with no penetrations whatsoever, and they are excluded from this part of the study, as were all teeth where adjoining caries, fractures, etc. left doubt as to the accuracy of our observations. Four hundred and six teeth remained. Before sixteen, 33% presented a circumferential groove of equal depth on all surfaces (=), 42% an Area of Deepest Penetration on the buccal area (m-bu, bu, and d-bu), 20% on the lingual areas, and rarely directly on the mesial and distal surfaces. After sixteen there is a constantly decreasing per cent of teeth on which there is no Area of Deepest Penetration (=), until after fifty years only 2% fail to show this characteristic. The predilection for the buccal areas continues through life, being 52% by twenty-five, and over 60% from there on. The lingual is the area of second choice for this deep penetration, running from 20-26% throughout life, with the mesial and distal surfaces sharing the remainder. The mesio-buccal surface (m-bu) is consistently the most frequent site of these deepest penetrations after sixteen years of age.

In *Graph 4 A* the teeth were subdivided into Tooth Groups, to determine any predilection for certain surfaces of specific anatomical groups of teeth as the site of the Area of Deepest Penetration. Surprisingly, 80% of the bicuspids showed these areas on the buccal as compared to 46-59% for the other teeth. Clinical memory tells me that this is the most common site of early cervical hypersensitiveness. Another surprise was to find 39% of the cuspids presented these deeper areas on the lingual surfaces, as compared to an average for this surface of only 14-23% on other teeth.

In short, the Area of Deepest Penetration can occur on any surface of any tooth,



Graph 3. The Area of Deepest Penetration (A) and The Area of Lesser Penetration (B) expressed in the per cent of times each occurred on a specific tooth surface when the teeth are divided into age groups. bu—labial/buccal; li—palatal/lingual; m—mesial; d—distal; =— depth of penetration equal on all surfaces on A (i. e. no Area of Deepest Penetration) and on B, equal on all surfaces but those already recorded on A as having an Area of Deepest Penetration (i. e. having no Area of Lesser Penetration). See text for discussion.

but is found most commonly on the mesio-buccal surface, next most commonly directly on the buccal surface, and next most commonly on the disto-buccal surface, except in cuspid teeth, where it is found at least as often on the lingual surfaces as on the buccal.

Graphs 3 B and 4 B present the same observations, but on the Area of Lesser Penetration—that area of deterioration presenting less depth of softening than the Area of Deepest Penetration, but more than that found on the other surfaces. Before sixteen years the presence of this characteristic is not too common (*Graph 3 B*), 62% of the teeth having an Area of Deepest Penetration failing to show an Area of Lesser Penetration. As the patient grows older, however, the incidence of this lesion increases until it is present in 71% of the teeth after fifty years of age. A glance at either *Graph 3 B* or *4 B* shows its preference for the lingual surfaces, in contradistinction to the preference of the Area of Deepest Penetration for the buccal surfaces. Again the bicuspids and cuspids seem to vary slightly from the general pattern (*Graph 4 B*) with a 33% incidence of this secondary deep penetration on the lingual of the bicuspids and an equal incidence on buccal and lingual of the cuspids (which had an unusual frequency of occurrence of the Area of Deepest Penetration on the lingual).

To summarize our findings on the variations in the location and in the depth of the deepest penetrations, it may be said in general that in childhood, when measurable penetration into the tooth is present, about 33% of the teeth will present equal penetrations all around the tooth, 42% deeper softenings in the buccal area, and 24% on the lingual, mesial, and distal. From then on through life there will be fewer and fewer teeth in which the penetrations are equal all around the tooth, more and more teeth in which one area is softened more than the rest, and an ever increasing frequency in which the buccal area is the site of this greatest softening. Secondary

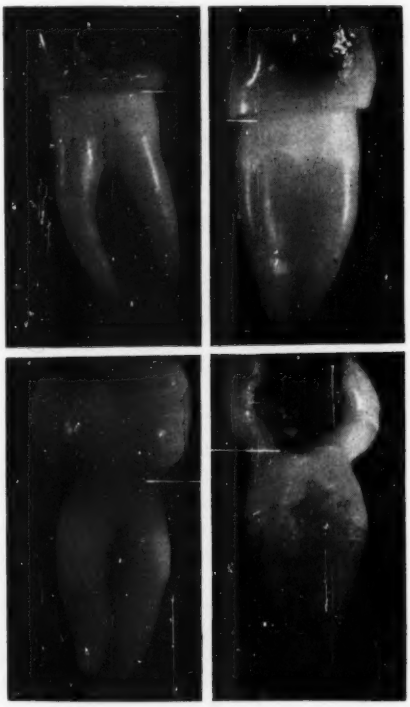
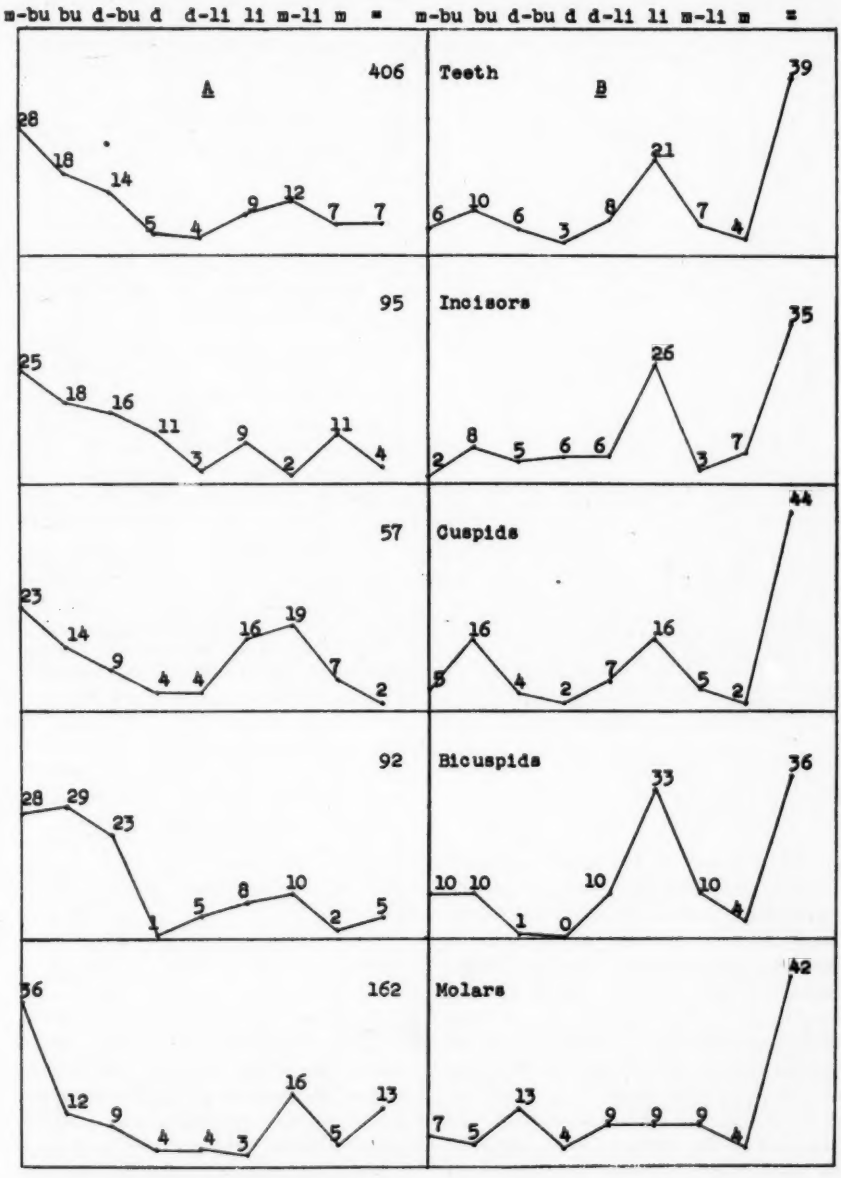


Fig. 1. The Line of Deepest Penetration.

areas of penetration (The Area of Lesser Penetration) will not be found in 62% of the teeth in childhood, but will be found with ever increasing frequency with the passage of the years, though even after fifty, 29% of the teeth will still fail to show this characteristic. When present, the lingual is its favored site.

THE LOSS OF TOOTH SUBSTANCE AT THE BOTTOM OF THE PATHOLOGIC CREVICE

At the bottom of the pathologic crevice, the loss of tooth was very small and it was impossible to be sufficiently accurate in relocating the point of original measurement to feel justified in presenting our findings in graph form. In general, however, the penetration at the bottom of the crevice is deepest in youth, when the clinging attached fibers of the periodontal ligament



Graph 4. The Area of Deepest Penetration (A) and The Area of Lesser Penetration (B) expressed in the per cent of times each appeared on a specific surface when the teeth are separated into anatomic groups. bu—labial/buccal; li—palatal/lingual; m—mesial; d—distal; = depth of penetration equal on all surfaces on A (i. e. no Area of Deepest Penetration) and on B, equal on all surfaces but those already recorded on A as having an Area of Deepest Penetration (i. e. having no Area of Lesser Penetration). See text for discussion.

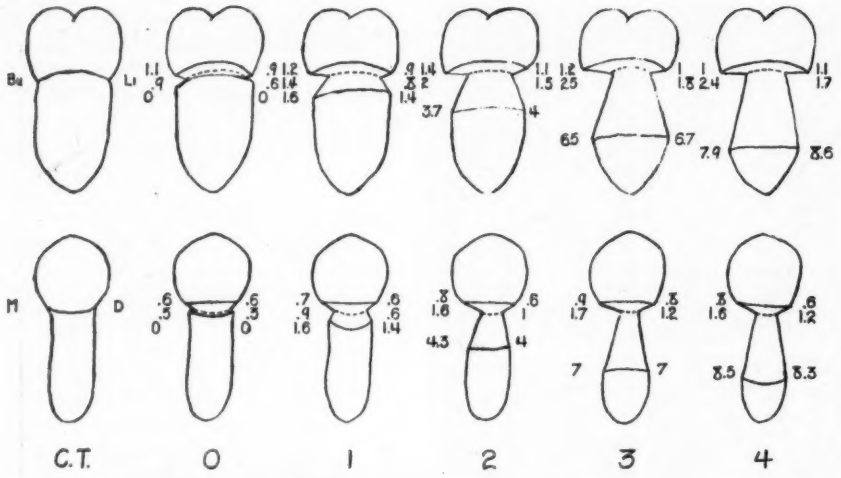


Diagram 1. THE PROGRESSIVE DEEPENING OF THE CHARACTERISTIC PATTERN, when grouped according to the depth of the pathologic crevice. The upper row represents the mesial aspect, and the lower row the buccal aspect of a Composite Tooth whose measurements are the averages of the teeth used in the study. (Drawn to scale 2.5 ×).
C.T.—Composite Tooth. 0—No pathologic Crevice (clinging attached fibers at the cemento-enamel junction). (51 teeth). 1—Clinging periodontal fibers apical to cemento-enamel junction, but less than ¼ the distance from the cemento-enamel junction to the apex. (191 teeth). 2—Fibers over ¼ but ½ or less the distance from the cemento-enamel junction to the apex. (133 teeth) 3—Fibers over ½ but ¾ or less the distance from the cemento-enamel junction to the apex. (82 teeth). 4—Fibers over ¾ the distance from the cemento-enamel junction to the apex. (29 teeth).
The top figure in each group represents the average loss of enamel at the cemento-enamel junction. The middle figure represents the average depth of the penetration at the Line of Deepest Penetration (original cemento-enamel junction).
The bottom figure is the average depth of the pathologic crevice before curettage when measured from the cemento-enamel junction to the remaining attached fibers.
(The picture is approximately the same when the teeth are divided into groups of 0-15 years, 16-25 years, 26-35 years, 36-50 years, and over 50 years.)

are still attached to the cemento-enamel junction. Then, as the bottom of the crevice exposes the cementum with the advancing years,[5] there are fewer and fewer teeth presenting a grossly measurable loss of tooth adjacent to the tissue detachment, until in late life, approximately 50% seem to present a measurable loss; 50% do not. When present, the amount of loss is about the same through life, being .5 to 1 mm in depth. As in our other observations, the frequency of this finding and the depth of the loss is slightly greater bucco-lingually than mesio-distally.

THE PROGRESSIVENESS OF THE LESION

Diagram 1 illustrates the progressive severity of the softening. The teeth are subdivided into groups according to the depth of the pathologic crevice, which division, as pointed out before, roughly coincides with the age groupings. They illustrate: 1. The unchanging amount of enamel loss through life (top row of figures); 2. The constancy of the location of the Line of Deepest Penetration at the original site of the cemento-enamel junction; 3. The increasing depth of the penetration at the cemento-enamel junction with increasing depth of the pathologic crevice (middle row of figures); 4. The almost constant pattern in which the penetration is deepest on the buccal (Bu), next deepest on the lingual (Li), and least on the proximal surfaces (M & D); 5. The characteristic sharp ledge formation from

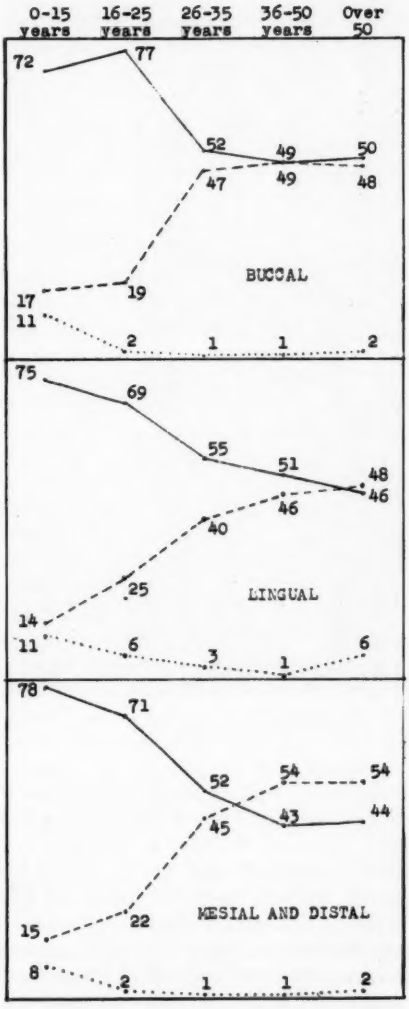
the Line of Deepest Penetration to the New Enamel Line; 6. The almost as sharp return to the original root surface in teeth with little or no pathologic crevice (0) to the long tapering slope of the apical side of the V-shaped groove in late life or deep crevice formation (4). The legend explains in detail.

RADIOGRAPHIC EVIDENCE

In the general observations section of this study[1] we described the relationship between "normal cervical radiolucency" as seen on the x-ray negative (Raper[6]) and the characteristic pattern of the lesion. Such radiolucencies were visible in over 90% of the extracted teeth studied, and in defense of the theory of normal cervical radiolucency it must be noted that 11% showed these characteristic darkenings at the cemento-enamel junction without any corresponding loss of structure during curettage. Neither did the x-ray always show the same degree of penetration as the post-curettage observations.

To estimate the relationship, the x-rays taken before curettage and the pattern formed by these penetrations after curettage were carefully compared and charted. The results are to be seen in *Graph 5*. Thus, in early life we were able to see the approximate depth of the penetration in some 70-75% of teeth, but after thirty-five years we could not tell as accurately, about half of them having more penetration than one would have judged in advance. Rarely did the x-ray show more penetration than was actually present.

With such indeterminate results, our findings will be of little help clinically. Even on these extracted teeth, where ideal conditions could be determined and duplicated and the teeth x-rayed both mesio-distally as well as bucco-lingually, and with no overshadowing of alveolar bone to add to the confusion, we were unable to forecast our results consistently enough to justify a prediction as to the depth of the penetration. Surely we could not do as well under the adverse conditions in the mouth.



Graph 5. X-ray Findings Compared to Post-Curettage Penetrations. The teeth are divided into Age Groups and the observations tabulated in per cent. ———— Depth of Penetration approximately the same as shown in the x-ray negative; — — — — Depth of Penetration more than shown; ..... Depth less than shown.

In practice, we have found it justifiable to expect rather deep losses of dentin when such radiolucencies are markedly visible; not justifiable to expect no loss of dentin if they are not.

No attempt was made to correlate the amount of loss on the x-ray with age or depth of the pathologic crevice. Madan and Brudevold,[7] in a study inspired by the author's observations and in consultation with him in its early stages, noted increasing incidence and extent of these cervical radiolucencies with age, particularly in the 40-50 years of age group—an interesting confirmation of our observations of the increasing severity of the depth of the penetrations after 36 years of age. They concluded, like Raper, that these radiolucencies could be duplicated by changing the angle of the passage of the rays through different anatomical curvatures of the root. Such a conclusion is not justified, however, unless one starts with a tooth in which there are no pathologic softenings—a condition rarely present in the age groupings they used. If these cervical radiolucencies appeared and disappeared by changing the angle of the rays, and then the tooth were curetted as in our studies, and following curettage it was found that no corresponding softening existed, then, and only then would one be justified in stating that these radiolucencies are *only* the result of variations in anatomical thicknesses and curves. For example, if one were to cut a wedge shape notch in a lead pencil, and the pencil be held between a bright light and a white sheet of paper, the shadow of the notch may be made to come and go at will by merely rotating the pencil, from the full depth of the notch through all gradations to complete invisibility. Most of these penetrations being on the buccal or lingual, and deeper on one surface than on the others, much the same situation would exist when teeth with such a lesion were x-rayed in the mouth, or in a skull, as in their study.

As mentioned above, radiolucencies are present in young and even in unerupted teeth, but the author favors the concept of an area of softening being present before the fibers of the periodontal ligament are even detached from the root—evidence of which was found in the above studies of the depth of the deteriorations in young teeth (*Graph 1 A 0-15 yrs; B - 0*). Studies now being followed seem to be presenting additional evidence that these softenings really are decalcifications and that they are present in advance of the loss of the attachment of the periodontal ligament.

DISCUSSION

In studying these findings, certain questions arise.

Is this softening pathologic? Probably "yes," for if it were the normal it would be present in youth and would not increase in incidence and severity with the passage of the years and the increasing depth of the pathologic crevice; neither would it be greater on one part of a tooth than on another surface of the same tooth; nor would these "Areas of Deepest Penetration" become more common as well as more severe with time; nor would "Areas of Lesser Penetration" develop.

Can the softening be explained as a normal deterioration of tooth structure as it is increasingly exposed to the contents of the mouth over the years? Probably "no," for very slight penetrations were found on sufficient teeth in late life to negate this thought. Then, there were some teeth in early life with extreme softening, indicating that the softening process can be rapid—more rapid than could be accounted for by the process of growing old. And, the "Areas of Deepest Penetration" would not exist; the deteriorations would be equal on areas of equal exposure.

What causes the softening? Inasmuch as the greatest area of deterioration is on the external surface of the tooth and as the softening does not reach the pulp except in extreme cases, we can assume that the attacking agent comes from the outside—not from the pulp. Studies now in progress indicate that decalcification is at least one factor in the change in the character of the tooth substance. We are all acquainted with "senile decay" in the cervical area in older patients. Is it difficult to imagine a "precarious" softening in these areas, such as we find with excavators in the deeper parts

of cavities after the visible caries has been removed—a state of affairs in which the tooth structure has physically softened but looks the same as the adjoining unchanged surfaces? We do not know that these softenings are the early stages of caries, but we do know, clinically, that a failure to *completely* remove these softenings frequently results in dental caries within a comparatively short period following collapse of the previously inflamed gingivae, whereas areas in which the softened tooth structure is successfully removed rarely become carious.

What are the softenings? They are not resorptions, at least in the usual sense of the term. These lesions are not a visible removal of the calcified tissues and their matrix, an "eating-away" of previously calcified tissue; they are not visible and have only been demonstrated by (1) curettage of the root surface; (2) radiographic confirmation of the presence of the lesion on these curetted teeth; and (3) their permeability to radioactive solutions. They do not show in either decalcified or ground sections of teeth prepared by the usual histologic technics.

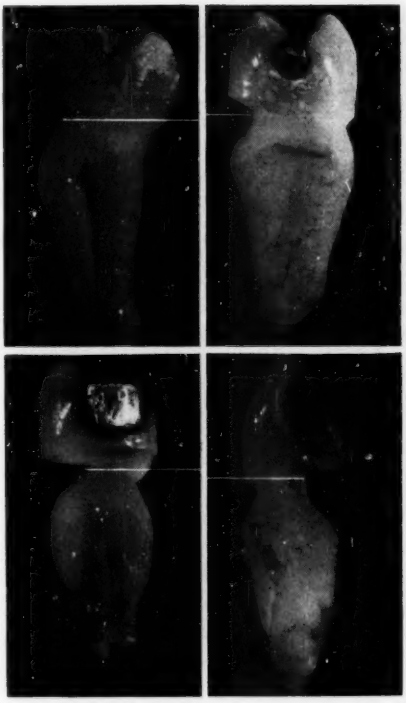


Fig. 2. The Area of Deepest Penetration.

Cahn[8] did demonstrate an enlargement of the dentinal tubules in the cervical area of pyorrhetic teeth and Bass[9] has described a peculiar granular condition found only in and under cementum exposed to a pathologic crevice and never in wedge shape abrasions, and which can only be demonstrated by a different technic of preparing the tooth for histologic examination.

Wainwright's studies[10] are the only really confirmatory evidence of the lesion we know. He has demonstrated on several occasions that radioactive solutions are rapidly assimilated by these changed areas in human teeth in a pattern identical to that of the lesions described in these studies—slight in the teeth of the young to very deep in the old long-exposed root surfaces —deeper in some areas than in other corresponding areas of the same tooth—even undermining the enamel at the cementoenamel junction and following the expected path of the dentinal tubules as we have described the lesion above. All of this occurs, of course, with no penetration of the normal tooth structure.

Hopewell-Smith[11] recognized what is possibly a late stage, but was more interested in the mechanism of the removal of the roots of deciduous teeth and the apices of abscessed teeth, than in the leathery softening in the cervical area of old cats' teeth, through which he was able to push a needle with ease. He did point out the possible mechanism of the formation of these softenings, by his description of the ability of granulation tissue to destroy bone and teeth, and was sure nature's methods are the same for teeth as for bone—"odontolysis" and "osteolysis," but his specimens were a visible removal of calcified tissues, similar to the case history in the human

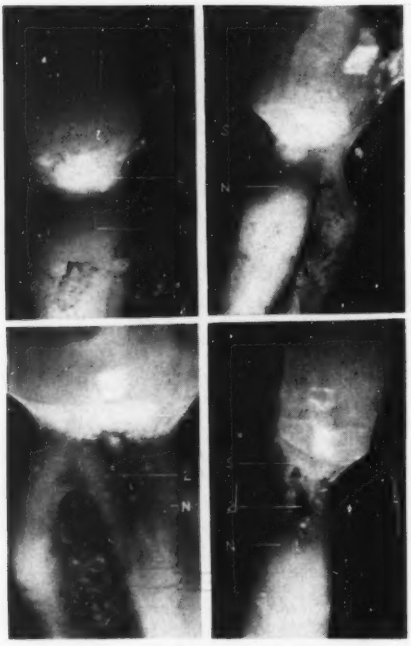


Fig. 3. Sclerotic and Secondary Dentin in the Area of Deepest Penetration.
S—Sclerotic (Opaque) Dentin. 2—Secondary Dentin. N—Normal Dentin.

presented by Mueller.[12]

One can easily imagine that there might have been a period of softening, before these visible resorptions, and McCall[13] did believe the inflammatory exudate beneath the inflammed gingival margin to be of sufficient acidity to cause surface roughening of the enamel and a more rapid dissolution of the root surface. Studies now in progress do seem to indicate that such a decalcification does take place, not only *after* the detachment of the periodontal ligament as visualized by McCall, but also *before* such detachment as well.

Weinmann and Sicher,[14] however, tell us that decalcification of bone would entail a considerable rise in local acidity and that the removal of bone entails the simultaneous disappearance of the organic and inorganic components, and that resorption starts with the removal of the inorganic components. They also tell us that where necrotic bone loses its connection with living connective tissue, resorption becomes impossible.

Furthermore, Ham[15] tells us that there seems to be no biologic counterpart for the decalcification procedures employed in preparing bone (and tooth) for sectioning; that bone loss is best viewed by considering it a failure in bone maintenance; and that bone surfaces exposed to tissue fluid (not cells) soon develop the etched appearance characteristic of resorption. He also tells us that bone is removed all at once—not preceded by decalcification.

The tooth surface of these wedge shape lesions is sometimes etched,[2,3,4] it certainly has lost its source of maintenance when the periodontal ligament becomes detached, and it probably has had a long period of inadequate nourishment from circulatory interference that accompanies the periodontal lesion, preceding the actual detachment of the fibers. That the acidity of the contents of the pathologic crevice is sufficient to cause such deep decalcifications remains to be proven. We do not have a simultaneous removal of the organic and inorganic constituents—a visible loss of structure.

Instead, we have a surface that in many cases looks perfectly normal, or only slightly roughened; an area that is probably already partly declacified *before* the loss of attachment of the periodontal ligament; and a deeper and deeper area *after* the loss of the fibers of the periodontal ligament, when there is no longer living connective tissue in biologic association with the calcified structure. Thus, if we are justified in comparing the known changes in bone to similar changes in teeth, our present lesion is not resorption.

Rather, the process seems to be more closely related to dental caries, where we do have a decalcification, preceding or following a loss in the integrity of the organic matrix. Thus, the enamel adjacent to and beneath inflammed gingivae is softened—changes (or never becomes "matured")

from the uncuttable smoothness of healthy enamel to a crusty removable material[2] which seems to be in direct proportion to the degree of the inflammation. As in dental caries, the process seems to be slow while confined to the enamel, because of its greater density and lack of organic pathways. That the process is related to the presence of the gingivae is evidenced by the fact that the deteriorations on the enamel do not increase in incidence or severity with the passage of the years (Diagram 1 and References[2]); rather, they take place in childhood, while the gingivae are attached to or covering the enamel, and do not increase to any significant degree throughout the rest of life.

Then, like dental caries, when the process reaches the cemento-enamel junction, the cementum, and the dentin, the deteriorations become more rapid, and though the tooth surface is no longer in physiologic connection with the surrounding tissues, the process continues, and the longer it continues, the deeper the softenings become. All this is evidenced by the deepest line of penetration always coinciding with, but slightly apical to, the original cemento-enamel junction, in the direction followed by the dentinal tubules—the area exposed for the longest period—while the shallowest penetration into the tooth is to be found at the bottom of the pathologic crevice—the area most recently exposed to the deteriorating process.

What is this "process" of deterioration? Like dental caries, we do not know. All we can do is surmise that the two are closely related, for these softened areas do become carious—gingival third and cemento-enamel junction cavities, cavities to the gingival of proximal restorations, and (to be discussed in another article) smooth surface caries as well.

CONCLUSIONS

Statistical summation of the results of curetting 500 extracted teeth in the area of the pathologic crevice, show:

1. The characteristic wedge shape penetrations into the dentin to be a progressively increasing loss from almost no softening in youth to an alarming depth in later years, even to the point of pulp exposure and fracture in 38% after fifty years of age.

2. A general trend in which INCREASING AGE = INCREASING CREVICE DEPTH = INCREASING DEPTH OF PENETRATION.

3. The deepest penetrations to be on the buccal surfaces, with the mesio-buccal predominating, particularly on bicuspid teeth.

4. Secondary areas of deep penetration to be most common on the lingual.

5. The loss of tooth structure at the bottom of the pathologic crevice to be little more than the expected thickness of the cementum.

6. The loss of hardness to be visible, but not prognostic, on the x-ray negative.

A discussion of the probabilities of causation leads us to believe that the process is more closely related to dental caries than to the resorption of calcified tissue elsewhere in the body.

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(Continued on page 155)

Book Reviews

"Periodontology—Diagnosis and Treatment" by Frank E. Beube, D.D.S., Clinical Professor of Dentistry and Head of the Division of Periodontology, Columbia University School of Dental and Oral Surgery. First edition 752 pages with over 500 illustrations. Index. Price $15.00. New York: The Macmillan Co. 1953.

This timely and complete text was written to give specific descriptions of the various technics for the diagnosis and treatment of diseases affecting the periodontal structures. It is divided into twenty-six chapters with a foreword by Dr. Arthur H. Merritt.

The organization of the text is somewhat unusual in that the chapter on Examination Procedures does not appear until a considerable portion of clinical pathology and therapy has been described. Your reviewer knows that the problem of where to teach this vital aspect of periodontology is a knotty one. Logic requires that it be taught early but practice shows that much is lost by projecting examination and diagnosis before the student has learned what to look for. Beube has placed this chapter in the middle of the text and another chapter of Diagnosis of Oral lesions at the very end of the book. This placement is interesting and has much to recommend it.

The book is very definitely the product of the clinical teacher. Much attention is paid to step-by-step procedures in most technics with technical hints and careful description of the procedures involved. Dr. Beube's "Periodontology" is definitely a teaching text and not primarily a reference book.

In the chapters on Occlusion and Selective Tooth Grinding there are several ambiguities and passages which could be clarified. In some instances the text could be studied with greater ease if the before and after illustrations were in consecutive order. Figure 342 on page 486 illustrates a type of splint that does not provide adequate embrasures and is incompatible with periodontal health. The author fails to make a clear distinction between the lesion of occlusal traumatism and inflammatory processes of the soft tissues. In chapter 12 on Harmful Oral Habits and Compulsions, Figure 268 leaves the impression that gingival recession has been caused by occlusal trauma. Occlusal trauma is also listed as a possible etiologic factor in necrotizing ulcerative gingivitis.

The therapeutic range of the book is a wide one with good coverage of the newer technics in the field. When it is considered that there have been so many valuable contributions during the past five years this attempt is noteworthy.

In the section on Necrotizing Ulcerative Gingivitis somewhat more attention to differential diagnosis would be helpful. This particular syndrome is so replete with myth and fancy in the literature of the past that a particularly clear analysis of the subject would render a great service.

The illustrations used are excellent. The line drawings in particular are unusually clear and uncluttered by extraneous detail.

The book is directed primarily to the student and general practitioner—who should also be a student. Nevertheless it may be read with profit by everyone who is interested in the field. It is a distinct contribution to the literature of periodontology.

John Prichard

"Clinical Periodontology—The Periodontium in Health and Disease" by Irving Glickman, B.S., D.M.D., F.A.C.D., Professor of Oral Pathology and Periodontology and Director of the Division of Graduate and Postgraduate Studies, Tufts College Dental School; Associate Staff

(Stomatology), New England Center Hospital; Consultant in Periodontology, Forsyth Dental Infirmary for Children; Lecturer in Oral Pathology, Boston University School of Medicine, Boston Massachusetts. First edition 1,019 pages with 742 illustrations. Price $15.00. Philadelphia: W. B. Saunders Co. 1953.

This voluminous book is encyclopedic in scope and covers the subject of Periodontology exhaustively. The text is augmented by over 700 illustrations, of which the drawings are particularly effective and beautifully done. The coverage of the important aspects of the subject is comprehensive, and that of the allied fields such as hematology, dermatology, oral manifestations of systemic disease, etc., is more than adequate.

The book is well organized, being divided into 3 main sections:

1. The Tissues of the Periodontium—wherein the clinical and microscopic descriptions of these tissues are given.

2. Periodontal Disease—devoted to the theoretical aspects of the subject.

3. Clinical Management of Periodontal Disease.

These sections, in turn, are broken down into subsections, and then chapters, in such a way that the subject matter is effectively compartmentalized, making the Table of Contents a valuable aid in finding information. Such division separates the theoretical from the clinical phases of the subject, permitting the reader to find easily that aspect of Periodontology in which he might be interested.

The text of the book is written with considerable erudition, requiring the reader in some instances to have sufficient background to follow the discussions of the various theories and descriptions. Clinical and microscopic descriptions of lesions, tissue changes, and the norm are listed side by side in many chapters, successfully correlating the macroscopic with the microscopic, thus making for greater lucidity. Throughout the body of the text these clinical and microscopic descriptions continue to complement each other in all aspects of the subject.

At the end of some chapters, such as "The Periodontal Pocket," "Bone Loss in Periodontal Disease," and others, the author comments on the various theories presented, correlates and summarizes these in a way that brings the material into sharp focus. However, on occasion these discussions become argumentative and lead to one-sided conclusions. This is especially true in the clinical sections of the book. The subsection on "The Etiology of Periodontal Disease" is handled well, a chapter being devoted to each etiologic factor. Prognosis, one of the most difficult aspects of Periodontology, is presented in a way that will be helpful to those who have had little experience in treating periodontal disease, yet wish to evaluate cases to be treated.

The weakness in this book lies in the section on Clinical Management of Periodontal Disease. The author is given to undue optimism and over-simplification. An example of such optimism is expressed in the description of the results of subgingival curettage in the gingival and infrabony pockets, as well as the gingivectomy technic of the gingival and infrabony pockets. The author gives the impression that reattachment is a rather common outcome of treatment, since he attempts it in each of the above operations. For instance, the composite retouched photomicrographs of Fig. 575, illustrating what may occur after scaling and curettage of the gingival type pocket, shows the epithelium and connective tissue attached at a higher level coronally. The caption reads "Composite illustration of various stages in pocket *eradication* using scaling and curettage." The finality of such an illustration and caption may be misleading.

However, earlier in the book the following statement is made: "It is the opinion of the author that if the epithelial attachment and the epithelial lining of the pocket are removed, and the root surface is

thoroughly debrided of all degenerated and foreign material, some degree of 'reattachment' would occur above the previous area of attachment of the epithelial attachment. The level to which this must reach in a coronal direction before one is justified in categorically stating that 'reattachment' occurs or does not occur following periodontal treatment is a moot point."

In reattachment attempts most clinicians and investigators agree that of several prerequisites for success, two are of great importance: first, the elimination of inflammation prior to the attempt; and second, the maximum protection of the blood clot. Dr. Glickman's gingivectomy technic for the eradication of the infrabony pocket is at variance with this, since it does not include prior scaling or curettage to eliminate or minimize the inflammation, and the incisions made as illustrated would result in a blood clot extensively exposed on the coronal and buccal surfaces of the wound. The application of a pack, no matter how delicately placed, is apt to dislodge the clot. The movement of the pack during the seven days it is left in place is likely to be detrimental to the result desired. The instrumentation for this technic is feasible for infrabony pockets on the buccal and lingual surfaces, but would appear to offer difficulties interproximally in many cases.

The gingivectomy technic for the eradication of periodontal pockets as described in the text, and illustrated by drawings, is an over-simplification of what the clinician must do in most instances.

The outline of the various depths of the pockets, and the "discontinuous incision" in Fig. 578 is adequate if the pocket morphologies permitted this, but generally speaking the morphological variations are too numerous to permit such a simple incision.

The author takes the stand that treatment should be either scaling and curettage or gingivectomy. He states that "limitation of clinical practice to 'scaling and curettage' alone, or adherence to a 'scaling and curettage first' clinical philosophy, are as ill-advised as the use of gingivectomy technic for all periodontal disturbances. Experimentation with scaling and curettage as a precursor to the gingivectomy treatment is contraindicated for the following reasons:"—and he presents reasons which are not valid. The various other factors discussed prior to these statements make this "either or" concept an oversimplification of the use of periodontal technics.

Throughout the book the biographical references are sweeping in scope, but in the chapters on clinical management several significant contributions on treatment have been excluded, and others of much less significance were included.

An excellent chapter on "Occluso-Rehabilitation" was written by Dr. L. A. Cohen, and is included in the section on Clinical Management of Periodontal Disease.

This volume is an important addition to the periodontal literature, and an excellent work for reference. It belongs in the library of every individual interested in Periodontology because of its wealth of information, some part of which will be valuable to every reader.

Nathan Friedman,
Beverly Hills, California

Periodontia: A Study of the Histology, Physiology, and Pathology of the Periodontium and the Treatment of Its Diseases. By Henry M. Goldman, D.M.D., director of the Riesman Dental Clinic, Beth Israel Hospital, Boston, Massachusetts; postgraduate faculty, School of Dentistry, University of Pennsylvania; and lecturer, Columbia University. Third edition. 790 pages with 525 illustrations. Price $16.00. St. Louis: The C. V. Mosby Company, 1953.

This text of almost 800 pages gives some indication of the exhaustive work in the preparation of Goldman's third edition. Though the number of pages are many, the book does not show any attempt at padding as the material is presented in a detailed manner. The bibliography is complete yet

not voluminous. Classic photographic illustrations and drawings are used to aid in a fuller understanding of the subject.

The book starts with the sciences related to the periodontium. Following in proper sequence are the various etiologic factors with the necessary suggestions for treatment. The chapter on scaling and instrumentation, which is basic in the treatment of periodontal disease, graphically illustrates the instruments to be used, the finger rests to be employed and the actual application to the tooth surface. The sharpening of these instruments is also described in this chapter. Other methods of treatment are likewise illustrated step by step with good drawings or photographs.

In reviewing this book in the Illinois State Dental Journal recently, Dr. Leo M. Streenby said:

"The usefulness of this book on periodontology is exemplified by the fact that there have been three editions of it in one decade. In a sense, however, this is a new book. Many changes have occurred in our knowledge of the etiology, pathogenesis, and treatment of periodontal diseases since the last edition. Dr. Goldman has completely reorganized and rewritten his book to include these changes. Emphasis is placed on those aspects of periodontology which are of most import to the general practitioner, i. e., the etiology, diagnosis, and techniques of therapy. These are fully described and correlated with our basic knowledge concerning the anatomy, histophysiology, and pathology of the periodontium."

"Dr. Goldman defines occlusal trauma as "that force or forces caused by mandibular movement—capable of producing pathologic changes in the periodontium." The changes produced, he termed "periodontal traumatism." Yet, the author violates his own definitions when in the chapter on "experimental pathology" he states that the wedging action of a needle implanted in the periodontal membrane causes the effects of "occlusal traumatisms."

"Another point of confusion is created in the chapter on gingivectomy, where the terms 'initial incision and initial cut' are used without clear-cut differentiation. If they are the same, then why does Fig. 318 (p. 459) show two different types of instruments, one which 'is used for the initial incision' and the other for 'initial cuts.'"

"The reviewer missed a discussion of the temporomandibular joint. Furthermore, the section dealing with calculus formation, even though an addition to this edition, is far from adequate. This latter fault is shared by most of the other textbooks on periodontia.

"The reproduction of the clinical photographs is not uniformly excellent, and in some instances one feels that the substitution or addition of diagrammatic sketches might substantially aid the text.

"These few critical remarks should not, however, detract from the merits of the book. This is a good book. It is indeed one of the best books written on the subject of periodontics today. Not only is it of value to the student who wishes to learn the basic fundamentals of periodontology, but also to the busy general practitioner who also wants to improve his technics of therapy, and to the specialist in the field who wants to re-evaluate and re-investigate many of the problems in this rapidly expanding field."

Anyone, whether he is a student, teacher, general practitioner or periodontist, will profit by having this text in his library.

Henry M. Swenson

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(Continued from page 137)

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Report on the Thirteenth Congress of the ARPA Internationale (Association for periodontal research)

by RAOUL H. BLANQUIE, D.D.S., *San Francisco, Calif.*

THE Thirteenth Congress of the ARPA Internationale was held July 13-19, 1953 at Geneva, Switzerland and attended by 250 members and participants from 26 different countries. This body was organized in 1932 at Frankfort-on-Main by the following well-known periodontists: A. J. Held and Rene Jaccard of Switzerland, Otto Loos and Oskar Weski of Germany and Charles Hulin of France. The ARPA is composed of national groups throughout the world and publishes their research contributions and activities in a journal entitled "Paradentologie." The following are the main objectives of the ARPA:

a. Develop scientific research of periodontal diseases.

b. Promote a close collaboration between dentists and physicians.

c. To bring about, on an international plan, the exchange of ideas and observations relative to periodontal diseases by means of meetings and by the publication of papers or periodicals.

d. Coordinate the efforts of national ARPA groups and of individual members or associates.

Each congress features certain specific subjects in periodontia and at Geneva this year, the following questions were discussed in three separate symposia:

a. Periodontal disease in children and adolescents.

b. Reactivation therapies.

c. Social aspect of periodontal diseases.

F. M. Pucci of Uruguay presented the following in the summary to his paper on "Precocious periodontal diseases."

1. The child undergoes physiological and biological changes that are entirely individual. The vital growth forces, the active cellular renewal, and the biochemical characteristics of the tissues and organs give, so far as pathological conditions are concerned, an intense response with prompt recovery. Acute processes predominate and chronic regressive and degenerative ones are not seen. The adolescent is subject to pathological peculiarities and is influenced by hormonal secretion and the final growth impetus. The young adult, during his adaptation to adult physiology is prone to chronic, atrophic, regressive and degenerative processes.

2. In precocious periodontal diseases the following can be observed:

GINGIVITIS—simple, edematous, fibrous, Vincent's, catarrhal and herpetic stomatitis.

PERIODONTITIS—When edematous gingivitis persists it tends towards periodontitis, especially during the last stages of puberty and in young adolescents. In children, periodontitis caused by alveolar atrophy around the temporary teeth may exist.

PERIODONTOSIS — Between adolescence and adulthood, systemic, hereditary and organically predisposing factors cause regressive and degenerative changes to prevail. In children with a balanced diet and sound ways of living, gingivosis, periodontosis and alveolar atrophies are not observed. Such processes arise in cases of malnutrition, endocrine and metabolic disturbances, either known or unknown, just like the different types of osteopathy.

Precocious atrophy of the marginal periodontium—we find these under three forms:

a. Continuous destruction of alveolar crests of early and late childhood.

b. Juvenile atrophy (13 to 18 years):

Advanced alveolo-gingival vestibular resorption, the rest of the periodontium being normal (due to gonadal action).

Pre-senile atrophy (beginning at 18 or 20 years): plastic and even throughout the contour of the marginal periodontium, progressing slowly with age.

Osteopathy with a precious effect on periodontium — Osteogenesis imperfecta, osteopetrosis, cleidocranial dyostosis, generalized osteitis fibrosa cystica. Hand-Schüler-Christian and Gaucher diseases, eosinophilic granuloma and other diseases related to endocrine dysfunction and avitaminosis.

3. The 25 statistics published show that periodontal lesions and their severity increase with age, having as principal etiological factors: defects of dental eruption, malnutrition, bad hygiene, abnormal occlusion, geographic, economic and social conditions.

J. J. Holst of Copenhagen, Denmark, defined Reactivation Therapy as those measures that tend to influence favorably the local tissue reaction upon the treatment of periodontal disorders.

A. J. Held of Geneva, Switzerland, stated that general reactivation, as well as local, must bring into action all means susceptible of improving local trophic conditions, that is to say mètabolic exchanges: by bringing about necessary factors to cellular combustion and the building of new elements destined to replace those that have been used up and also the elimination of the final products of this combustion and wearing out process. The application of correct quantity and proportions of elements of minerals, vitamins and oxygen constitutes the fundamental therapy of reactivation.

Many other subjects in periodontia were presented in the form of papers, motion pictures and clinics. French, English and German are the official languages and following each paper a summary in the other two languages was made.

This meeting accomplished many things such as acting as a clearing house for the latest developments in periodontia by bringing together some of the leading authorities from the four corners of the world. Not the least was the feeling of good-will and friendship that it engendered between so many different nationalities.

The six days of the meeting were replete with action and with time out only to enjoy the generous hospitality of the members of the local committee who, each day, invited different groups of visiting members to have lunch or dinner in their homes. Tours in and about the city were arranged for the ladies.

The next ARPA meeting will be held in Venice, Italy, in 1955 and the Italian ARPA has already started plans towards making it most outstanding. American periodontists are most cordially invited to attend and all doing so can be assured in advance of a most interesting and worthwhile vacation.

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(Continued from page 149)

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THE OASI AND THE DENTIST

Since the Federal Old Age Insurance program has been the subject of debate at several recent meetings of the American Dental Association, a study of just what it would mean to the individual member, is a matter of no little importance to all concerned. The need of such understanding is increased by the fact that the present Congress is being asked to consider the inclusion of several of the professions in OASI, including dentistry, medicine, and law, plus architects, accountants, and clergymen among other self employed groups. The necessity of an informed membership cannot, therefore, be overemphasized.

One will need to understand first of all that the self employed individual will be obliged, *under present law,* to pay 50% more than the wage earner or salaried employee: that once adopted by a profession, *compulsory participation is required by every member.* No one can withdraw. Neither can future generations who will have had no voice in making the law.

One needs also to understand that the so called contribution to OASI will be added to ones income tax on a graduated scale, increasing to 4⅞% in 1970 on the first $3,600 of ones income, with the recommendation that it be increased to $4,200. This would mean that 15 million people would have more of their earnings taken into account by the program. This would mean that the participant at present rate would be required to pay $175.50 each year as an additional tax. Over a period of 40 years this would amount to over $7,000. When to this is added compound interest at 3% it would amount to a total in excess of $13,000.

After age 65 of both husband and wife, they would jointly receive $120.00 a month or $1,440 annually. Both would have to live to age 74 to get back the $13,000 or more it had cost them. If the insured should die before his 65th birthday, his estate would receive no part of what he had paid as a participant of OASI except $255 at death.

Unless changed by law, all would be subject to the following conditions:

1. The beneficiary would have to be 65 to receive $85 per month for life.
2. A wife must be 65 or more to receive $42 a month.
3. If a wife predeceases her husband after age 65 he would still receive only $85 a month.
4. If the husband predeceases his wife after age 65, she would receive $63 a month providing she is over 65.

All of the above payments are contingent upon ones not earning more than $75.00 a month by any employment in which he might engage. Under these conditions, the participant is precluded from engaging in a more remunerative activity without sacrificing his investment in OASI.—*A.H.M.*

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Announcements

THE ANNUAL SPRING SEMINAR OF THE AMERICAN ACADEMY OF PERIODONTOLOGY

The Annual Spring Seminar of the American Academy of Periodontology will be held in Ann Arbor, Michigan, April 19, 20 and 21, 1954. The program is as follows:

Monday, April 19, 1954:

8:30- 9:30 a.m.	Registration—Room 2004, W. K. Kellogg Foundation Institute
10:00-12:00	The Alarm Reaction and Adaptation Syndrome. Dr. Andre Robert, University of Montreal.
2:00- 4:30 p.m.	Significance of Alarm Reaction and Adaptation Syndrome in Periodontia. Reactions of the Oral Tissues in the Alarm Reaction and Adaptation Syndrome. Dr. Andre Robert.

Tuesday, April 20, 1954:

9:00-12:00 a.m.	Clinical Significance of ACTH and Cortisone and Its Application in Present Day Medical and Dental Practice. Dr. Stephan Fajans, University of Michigan Hospital.
2:00- 5:00 p.m.	The Development of the Periodontal Pocket. Dr. Jens Waerhaug, Oslo, Norway.

Wednesday, April 21, 1954:

9:30-12:00 a.m.	Conservative Management of the Periodontal Pocket. Dr. Frank Beube, Columbia University.
1:30- 3:00 p.m.	Surgical Management of Periodontal Pockets. Dr. Henry Goldman, Boston, Mass.

Anyone interested should get in touch with Dr. William R. Mann, University of Michigan, Ann Arbor, Michigan.

TENTATIVE PROGRAM AMERICAN ACADEMY OF PERIODONTOLOGY MEETING

Miami, Florida, November 4-6, 1954

Thursday, November 4, 1954

11:00 a.m.	Important Advances in Periodontal Research and Their Clinical Implications. Dr. Harold Ray, San Francisco, California.
PM—	Symposium: The Role of other Fields of Dentistry in the Prevention and Treatment of Periodontal Diseases.
2:00 p.m.	Physiology of Jaw Relations. Speaker to be selected.
2:30 p.m.	Operative Dentistry. Speaker to be selected.
3:00 p.m.	Orthodontia. Speaker to be selected.
3:30 p.m.	Fixed Bridge Prosthesis. Speaker to be selected.
4:00 p.m.	Removable Bridge Prosthesis. Speaker to be selected.

Friday, November 5, 1954

9:00 a.m. A Concept of the Epithelial Attachment and Gingival Pocket. Jens Waerhaug, Oslo, Norway.

9:30 a.m. A Concept of the Epithelial Attachment and Gingival Pocket. Speaker to be selected.

10:00 a.m. Physical Role of Calculus in Relation to the Epithelial Attachment and Gingival Pocket. Helmut Zander, Minneapolis, Minn.

10:30 a.m. Chemical Role of Calculus in Relation to the Epithelial Attachment and Gingival Pocket. Speaker to be selected.

11:00 a.m. Bacteria in the Epithelial Attachment and Gingival Pocket. Speaker to be selected.

11:30 a.m. Discussion of the Clinical Implications of Above. Speaker to be selected.

2:30 p.m. Selective Attendance Program:

A. Subgingival Curettage. Speaker to be selected.
B. Physiology of Connective Tissue. Speaker to be selected.
C. Management of Oral Manifestations of Systemic Disease. Speaker to be selected.
D. Periodontal Therapy in Systemic Disease. Speaker to be selected.

Saturday, November 6, 1954

9:00 a.m.-11:00 a.m. Round Table Discussion: Wound Healing and its Relation to Various Methods of Periodontal Surgery

9:00 a.m. 1. Physiological Aspects of Healing. Speaker to be selected.

9:15 a.m. 2. Histochemical Aspects of Healing. Speaker to be selected.

9:30 a.m. 3. Curettage. Speaker to be selected.

9:45 a.m. 4. Gingivectomy. Speaker to be selected.

10:00 a.m. 5. Flat Approach. Speaker to be selected.

10:15 a.m. 6. Gingivoplasty. Speaker to be selected.

10:30 a.m. Exchange of Questions between Experts on Panel.

10:45 a.m. General Discussion.

Clinics begin at 2:00 p.m.—Tolar Hamblen, Chairman

THE FIFTH ANNUAL BERKSHIRE CONFERENCE IN PERIODONTOLOGY

Tufts College Dental School Postgraduate Division will conduct an advanced conference in Periodontology and Oral Pathology under the direction of Dr. Irving Glickman and staff at the Cape Codder, a resort hotel in Falmouth, Mass., on Cape Cod, from June 20-24, 1954. The conference will consist of four days of lectures, seminars, and panel discussions regarding clinical problems by outstanding teachers and clinicians in dentistry, medicine, and their related fields. A special feature of the conference will be a clinico-pathological seminar with microscopes provided for all participants.

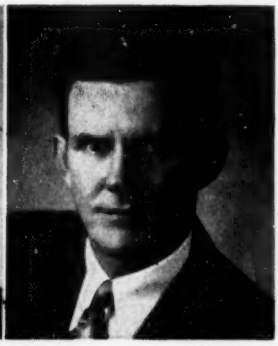
The curriculum is arranged so that mornings and evenings are devoted to professional activities with afternoons open for recreation. This is a conference for general practitioners as well as those interested in specialized practice.

Application may be obtained from Tufts College Dental School, 136 Harrison Avenue, Boston 11, Mass.

OFFICERS OF THE SOUTHERN ACADEMY OF PERIODONTOLOGY

Frank O. Alford, D.D.S.
President

Ernest L. Banks, D.D.S.
Secretary-Treasurer



John S. McKenzie, D.D.S.
President-Elect

NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

New York University College of Dentistry announces a two year full time postgraduate course in Periodontia and Oral Medicine in preparation for certification and teaching. One year full time, two year half time and four year half time courses also are offered, starting September 20th, 1954. The above courses are given by the Periodontia Faculty under the direction of Dr. Samuel Charles Miller. A variety of other courses are also available in this and all other phases of dentistry.

For information write: Secretary, Postgraduate Division, New York University College of Dentistry, 209 East 23rd Street, New York 10, New York.

PERIODONTOLOGY COURSES AT COLUMBIA UNIVERSITY

Four postgraduate courses in Periodontology are offered at Columbia University, School of Dental and Oral Surgery of the Faculty of Medicine.

A full-time program covering a period of two academic years and a half-time program covering a period of four academic years lead to a Certificate of Training in Periodontology. Correlation of the fundamental sciences to clinical practice is emphasized.

Of the two part-time courses, one course will be given one full day a week from September 22, 1954 to May 4, 1955; the second course will be given one half day each week from September 22, 1954 to May 4, 1955.

For further information write to Postgraduate Division, School of Dental and Oral Surgery, Columbia University, 630 West 168th Street, New York 32, N. Y.

CALIFORNIA ACADEMY OF PERIODONTOLOGY

Officers of the California Academy of Periodontology for 1954 are: President—Samuel Bleadon, D.D.S., 2827 Franklin St., San Francisco, Calif. Vice-President—R. Thomas Dunkin, D.D.S., 410 Twohy Bldg., San Jose, Calif. Recording Secretary—Howard J. Mallon, D.D.S., 2844 24th St., San Francisco, Calif. Financial Secretary—Elmer G. Smith, D.D.S., 1361 Park St., Alameda, Calif. Treasurer—Clarence E. Butler, D.D.S., 450 Sutter St., San Francisco, Calif.

COURSES IN PERIODONTICS

by Balint Orban, M.D., D.D.S.

HISTOPATHOLOGY OF THE SUPPORTING TISSUES	June 14-18, 1954
OCCLUSION	June 26-30, 1954
THEORY AND PRACTICE OF PERIODONTICS	July 2-6, 1954

The following courses will also be offered:

DISSECTION OF THE HEAD AND NECK — June 18, 19, 20, 1954

by Dr. Harry Sicher

TUMORS OF THE ORAL CAVITY — June 26-30, 1954

by Dr. Joseph P. Weinmann

Upon request, additional information and outlines of the courses will be given. Please address all communications to Dr. Balint Orban—629 N. Nevada Ave., Colorado Springs, Colorado.

COLORADO DENTAL FOUNDATION SEMINAR
JUNE 20-24, 1954

No details available, but can be obtained by writing the Foundation at 629 N. Nevada Ave., Colorado Springs, Colorado.

SPECIAL POSTGRADUATE COURSES OFFERED AT NEW YORK UNIVERSITY COLLEGE OF DENTISTRY DURING THE MONTH OF JUNE

Periodontia 123C	6/7/54-6/11/54	Daily	9-4
Periodontia 124	6/7/54-6/25/54	Daily	9-4
Pulp-Canal Therapy	6/7/54-6/18/54	Daily	9-4

These courses include lectures and clinic experiences in the many phases of theory and practice of dentistry.

A one-week special review course, Periodontia 123C is limited to students who have participated in other periodontia postgraduate courses.

For information write: Secretary, Postgraduate Division, New York University, College of Dentistry, 209 East 23rd Street, New York 10, N. Y.